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A MONOGRAPHIC STUDY OF SWEET-POTATO DISEASES AND THEIR CONTROL

BY

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INTRODUCTION

The sweet potato (*Ipomoea batatas* Poir.) ranks as one of the most valuable and dependable food crops in the tropical and semitropical world, and next to the potato (*Solanum tuberosum* L.) it is the most

[1] The writers acknowledge the assistance contributed in various ways by some of their coworkers. They wish to thank J. I. Lauritzen for the use of certain experimental data and W. A. Whitney and C. B. Sumner for help in the preparation and arrangement of the illustrations, reading of the manuscript, and verification of citations.

important vegetable in the United States. The sweet potato grows as a perennial in tropical countries, but not in the United States, where it has been developed and commercialized probably to a greater degree than in any other country. For this reason the crop has been adjusted to meet wider variations in cultural conditions than the natural habits of the plant would suggest were possible. In many tropical countries the sweet-potato planting is more or less permanent, the plants maintaining themselves for several years without being reset and without much cultivation.

There is very little accurate information on the origin of the sweet potato. Two theories are advanced: (1) That it is of Asiatic origin; and (2) that it originated in tropical America. De Candolle (*21*),[2] although not committing himself definitely, seems to be of the opinion that it is of tropical American origin. The same view is held by Groth (*59*), who states that the evidence presented warrants fully the conclusion that the sweet potato is a native of tropical America. According to Groth, Columbus discovered the sweet potato in Cuba on his first voyage to America.

The sweet potato is cultivated primarily for the fleshy root, which in some of the varieties often attains a weight of several pounds. The cells in the roots are gorged with starch, and a little sugar is also present.

An examination of the statistics reveals the fact that the acreage of sweet potatoes gradually increased in the United States from 1899 to and including 1922, showing that the crop is becoming of more economic importance among the food crops of the country. Following 1922, there was some decrease in the acreage. In 1899, 42,517,412 bushels (*195*) were produced in the United States, with a value of $19,869,840. In 1922, 109,394,000 bushels (*192*) were grown, which had an estimated total value of $84,295,000. More than 1,117,000 acres were planted in that year—probably the largest acreage in the history of the United States. No figures are available showing the relative value of this crop in countries outside the United States.

In this country the largest acreage of sweet potatoes is grown in the Southern States, although some of the Northern States (especially New Jersey, Delaware, Maryland, Iowa, Kansas, California, Illinois, Ohio, Washington, and Indiana) also produce a considerable quantity. Pennsylvania, Nebraska, Colorado, Utah, and a number of other States grow sweet potatoes for home consumption. The acreage by States in 1924 is indicated graphically by Figure 1.

FARM PRACTICES EMPLOYED IN GROWING SWEET POTATOES

In view of the relationship existing between the methods of handling the sweet-potato crop in the United States and the general prevalence of its diseases, a brief consideration of the farm practices employed is essential. In no country in the world is the sweet potato cultivated and handled as it is in the United States, and, so far as the writers are able to determine, the destructive diseases are not so generally present in any other country. This correlation

[2] Italic numbers in parentheses refer to "Literature cited," p. 108.

between the prevalence of the diseases and the methods of handling the crop probably is not accidental.

The sweet potato is grown in the United States as an annual; that is, a new crop is planted each year. This is obligatory, since sweet potatoes are very sensitive to frosts, and in no part of the United States will they survive the winter, with the possible exception of southern Florida. The roots that are to be used for seed purposes the following season are dug in the fall just preceding or immediately following the first frost and are stored during the winter. In the spring they are removed from storage and bedded in soil or sand, where young plants are produced for the initiation of the next crop. In many sections of the country it is the general practice to lay the seed potatoes in the bed about one-fourth to one-half inch apart and more or less parallel with one another. The

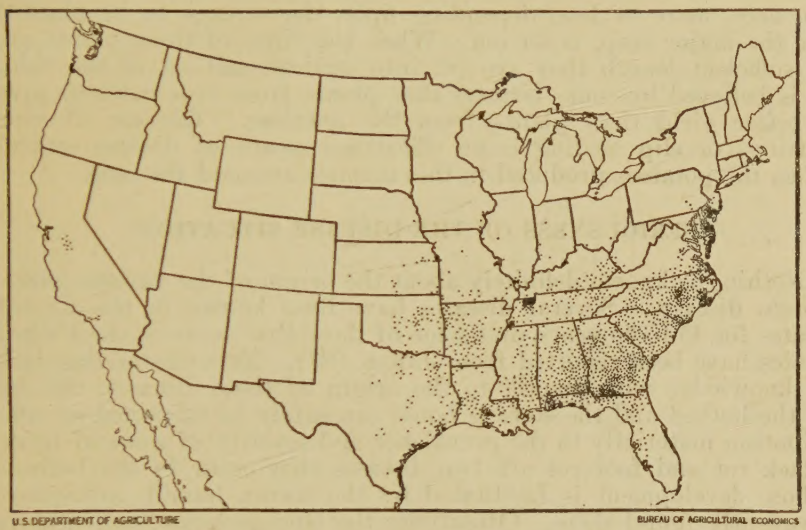


FIG. 1.—Outline map of the United States, showing by black dots the distribution of sweet-potato acreage in 1924. Each dot represents 20,000 bushels. (Data prepared by the Bureau of Agricultural Economics)

spacing of the potatoes in the bed not only permits the healthy development of the young sprouts but also, at least to some extent, prevents the spread of diseases, if any are present, from one potato to another. In other sections it is the practice among some growers to dump the potatoes into a ditch, level them off with a rake, and then cover them with soil. When the latter method is followed the potatoes are usually very thick in the bed, and the communication of black rot or other diseases from potato to potato or from one plant to another is very prevalent.

In the northern border of the sweet-potato belt hot water, steam, hot air, or fermenting manure are used as the sources of the heat supplied to the bed. The beds are frequently covered with canvas or with glass sash to retain the heat and moisture in the soil. In from three to five weeks the plants are large enough to pull from the mother potato and set in the field, where they are planted from

16 to 20 inches apart in rows about 3½ feet apart. In some sections the plants are set on ridges and in others they are not. They are cultivated about the same as any other crop that is set out in rows, usually by means of plows and cultivators.

In parts of the United States where the season is sufficiently long, another form of propagation, known as slip seeding or vine cutting, is sometimes practiced. This method of propagation consists of cutting the vines into sections from 6 to 12 inches in length and then planting each section in the ground to form a single hill. Vine cutting does not obviate entirely the necessity of bedding potatoes, but it does reduce considerably the quantity that must be bedded. In order to propagate by vine cuttings, especially where the plants may be killed prematurely by frost, the potatoes are bedded as early as weather conditions permit. The plants produced from the potatoes are set in the field, and the cuttings are made from them. An acre, more or less, depending upon the acreage to be planted for the major crop, is set out. When the vines of these plants are of sufficient length they are cut into sections and set in the field. It is believed by some farmers that plants from vine cuttings give a better yield than plants from the potatoes. The use of vine cuttings or slip seeding is an efficacious means of disease control when the potatoes produced in this manner are used for seed.

SERIOUSNESS OF THE DISEASE SITUATION

Nothing is known definitely about the origin of the various sweet-potato diseases. Several diseases have been known in the United States for 45 to 50 years, and some of those that occur in the United States have been reported from Japan (*99*). Notwithstanding lack of knowledge with respect to the origin of these diseases, the use of the hotbed and the storage house can safely be suspected of contributing materially to the prevalence and severity of some of them. Black rot and foot rot are two diseases that occur in the hotbeds whose development is facilitated by the warm, humid atmosphere frequently found there. Oftentimes the storage house, and usually the banks, provide a congenial environment for the propagation and transmission of these diseases. Such environmental conditions, coupled with the susceptibility of some varieties to certain diseases, partially explain the seriousness of the disease situation in the United States at the present time.

The losses sustained from the various diseases in field and hotbed can be only approximately estimated. The diseases in storage are perhaps of more economic importance than those in the field and hotbed, but here again it is not possible to give a very accurate statement of the losses. The estimated yields of sweet potatoes in the United States during 1926, according to Crops and Markets for December 25, 1926, was approximately 83,658,000 bushels (*193*). An estimated loss of the crop from sweet-potato diseases, both in the field and in storage, was obtained for a number of the States for 1925 by the plant disease survey.[3] The loss from field diseases in New Jersey in 1925, for example, was estimated at about 36 per

[3] UNITED STATES DEPARTMENT OF AGRICULTURE, BUREAU OF PLANT INDUSTRY. CROP LOSSES FROM PLANT DISEASES IN THE UNITED STATES IN 1925. U. S. Dept. Agr., Bur. Plant Indus. Plant Disease Rptr. Sup. 49 : 397–398. 1926. [Mimeographed.]

cent, or approximately 1,195,000 bushels, worth probably $1 a bushel. The estimated loss in storage was 4 per cent, or about 84,000 bushels, approximately equivalent to $84,000, which should be added to those losses already enumerated. This is the highest percentage of loss from field diseases reported from any State. In Arkansas there was a reported loss of about 8 per cent of the crop from field diseases and one of 30 per cent in storage. Alabama lost about 8.5 per cent in the field and 5 per cent in storage. In 1925 the average loss, including losses in the field and in storage, for all the States reporting was about 17.2 per cent, being equivalent to approximately $11,000,000 at the average price of $1 a bushel.

The fact must not be overlooked, however that the losses do not end with those obtaining in the hotbed, in the field, and in storage. Sweet potatoes must frequently be shipped long distances, and the losses in transportation as well as on the wholesale and retail markets, are sometimes very large. The reports of the market inspectors at some of the large shipping points, such as New York, Philadelphia, and Pittsburgh, frequently show losses in the cars ranging from a trace to 95 per cent. A careful examination of a number of reports shows that 15 to 25 per cent of the sweet potatoes that are inspected at the markets have decayed in the cars. Further losses may occur before the potatoes are consumed. In view of the losses that occur in the field, in storage, in transportation, and on the market, it is probable that not more than 60 per cent of the crop is actually consumed, the remaining 40 per cent being a total loss.

This monograph is prepared for the purpose of summarizing all the known available data on sweet-potato diseases. Much of the data submitted and many of the conclusions drawn are the results of work by the writers devoted almost exclusively to the diseases of this crop for a period of 16 years. The writers have also made liberal use of the investigations of other workers and have attempted to use their results impartially. This work is intended to give, as far as possible, an exhaustive survey of the sweet-potato diseases in the United States and in foreign countries.

A work of this kind, to be useful, must be so subdivided that correlated material can be brought under the same heading. The writers, with this thought in mind, have attempted to treat the subject of each disease under three heads: (1) An exhaustive survey, in so far as facts are available, covering the history of the disease, its geographical distribution, its economic importance, and its symptoms; (2) a discussion of the identity, pathogenicity, morphology, life history, and dissemination of the causal organism; and (3) the control of the disease.

DISEASES CAUSED BY FUNGI

MAJOR FIELD DISEASES

STEM ROT OR WILT

History

Stem rot, caused by *Fusarium batatatis* Wr. and *F. hyperoxysporum* Wr. and described by Halsted (*61*), seems to be the first common name given to this disease and the one by which many farmers know it at the present time. Since the name "stem rot" has become so

well established in the literature, it will be retained by the writers, although they realize that "wilt" would be more appropriate, for the reason that diseases of other crops caused by other species of Fusarium are generally referred to as wilt. Stem rot is sometimes called blue stem, cholera, yellow blight, wilt, and split stem.

Although it is of interest and frequently of some economic importance to know the place of origin of a disease, it is doubtful whether this information can be given definitely for any particular disease. The stem rot of sweet potatoes is believed to be indigenous to the United States. Reports indicate that the disease occurs in Japan (*99*), but this is the only country outside of the United States where it is known to be present. A considerable quantity of pathological material of sweet potatoes has been examined from tropical America and other foreign countries, but neither of the organisms causing stem rot has been isolated. It was especially searched for in Cuba, but it was not found there.

In regions where stem rot occurs no other hosts have been found for it under natural conditions. A number of plants other than the sweet potato, both cultivated and wild, among them several species of Ipomoea, have been artificially inoculated, but the disease was produced only in *Ipomoea hederacea* (*80*), a wild morning-glory. Other species of Ipomoea commonly found in sweet-potato fields and the cultivated plants generally grown with sweet potatoes or in rotation with them were tested for susceptibility to stem rot. It is therefore believed that the stem-rot organism has no other natural hosts with which it could have been introduced into this country.

The sweet potato is supposed to be of tropical American origin, but the stem rot has been found neither on plants there nor on material obtained from that source. Furthermore, many of the varieties of sweet potatoes grown in the southern part of the United States, with a few exceptions, are only slightly susceptible, even when subjected to artificial infection. On the other hand, the varieties most susceptible to the disease, with one or two exceptions, are those grown in the northern range of the sweet-potato belt, such as Yellow Jersey, Big-Stem Jersey, and Red Jersey. Whether or not the disease originated in these varieties will always remain a matter of speculation. The disease is certainly worse in the varieties that are restricted largely to the northern territory and in the Nancy Hall and Porto Rico varieties, which are grown more or less throughout all the Southern States. If it is suspected that the origin is along the North Atlantic seaboard, its appearance in the Southern States, where it is less destructive, could be explained by the shipment of the northern varieties to the South for trial or cultivation.

The time of the first appearance of stem rot is as indefinite as the place of its origin. The first account of the disease, so far as the writers know, was published by Halsted (*61*) in 1890. In 1892 he (*62*) again discussed the disease as "eggplant stem rot" and concluded that the stem rot of eggplant and sweet potato was caused by the same fungus, *Nectria ipomoeae* Hals. In 1895 he (*64*) attributed the ring rot of sweet potatoes to this same organism. Although these reports constitute the first published account of stem rot so far as the writers have learned from the literature, there are good reasons to believe that the disease was known in New Jersey long before 1890.

For 10 to 12 years after Halsted published a description of the various sweet-potato diseases, little or no mention was made of their occurrence. In 1904 and again in 1911 (*163*) a rot of sweet potatoes, due to a Fusarium, was reported as common and destructive in Merced County, Calif. In 1905 and in subsequent reports Stevens (*165*) mentions a destructive wilt disease of sweet potatoes occurring in North Carolina and gave in one of his reports a brief description of the characteristic symptoms. Stevens suspected that the disease was caused by a species of Fusarium, which he isolated from some pathological material. Almost simultaneously, reports from other States indicated that stem rot occurred in them, but in most of the reports *Nectria ipomoeae* was suggested as the probable cause. Townsend (*190*), and later Wilcox (*207*), discussed stem rot of sweet potatoes, accepting Halsted's determination of the causal organism. In none of the brief and incomplete reports was mention made of the length of time the disease had been present. The nature of several of the reports made by pathologists throughout the country indicated that they had in mind the disease that is known at the present time as stem rot. It is inferred, however, that they were in doubt as to the true cause of the trouble, since they mention *Fusarium* sp. and not *N. ipomoeae*. In 1913 Harter and Field (*78*) published an abstract embodying the results of experiments which definitely proved that the disease was caused by a Fusarium and not by *N. ipomoeae*. Later work (*80*) by the same investigators demonstrated that two species of Fusarium may be responsible for the disease, both of which cause identical pathological symptoms. Taubenhaus (*177*) later confirmed the conclusions of Harter and Field that stem rot was not caused by *N. ipomoeae* but by a Fusarium.

Geographical Distribution and Economic Importance

Stem rot, so far as the writers are aware, occurs only in the United States and possibly in Japan (*99*), where it is described as being due to *Nectria ipomoeae*. The many reports of its occurrence and the numerous collections made by the writers show that it is very widely and generally distributed throughout the United States. The disease has been known in New Jersey since 1890. It was reported by Selby in Ohio in 1903, 1906, 1907, 1909, and 1911. R. E. Smith and S. S. Rogers reported stem rot from California in 1904 and 1910, respectively. J. T. Barrett reported it from Illinois in 1907 and H. W. Barre from South Carolina in 1908. In North Carolina it was reported by F. L. Stevens in 1905 and 1910, and in Maryland by J. B. S. Norton in 1910. Mel. T. Cook reported stem rot in Delaware in 1911 and 1912 and in subsequent reports. Some of the foregoing data were obtained from the unpublished reports made by the collaborators of the Office of Vegetable and Forage Diseases of the United States Department of Agriculture. It is believed that in most if not all cases the reports were made of the same disease that is known as stem rot at the present time, although *N. ipomoeae* was mentioned in some of them as the cause.

In 1914 Harter and Field (*80*) published the results of an extensive study of the stem-rot fungus from material collected in Mary-

land, Virginia, Delaware, and New Jersey, and also from material sent from Alabama. In 1915 the known distribution (*71*) was extended by collections in Ohio, Illinois, Missouri, Iowa, Kansas, Oklahoma, Arkansas, North Carolina, Georgia, and Mississippi, and later in Texas and Florida (*75*). S. H. Essary advised the writers orally of its common occurrence in Tennessee, and C. W. Edgerton reported it as prevalent in Louisiana in 1918. In 1917 and again in 1923 stem rot was found to be very prevalent in Orange, Los Angeles, Merced, and Stanislaus Counties, Calif. Typical symptoms of the disease were produced by inoculation experiments from cultures made from the material. The causal organism was studied both culturally and morphologically and identified as *Fusarium hyperoxysporum* Wr. In 1926 stem-rot specimens were collected at Kennewick, Wash., in the Jersey and Nancy Hall varieties, and at Greeley, Colo., thus extending the range of distribution considerably farther north. The disease apparently was introduced by seed potatoes from some of the States farther south.

The loss from stem rot can not be estimated except within wide limits. New infections occur during the entire growing period, so that the yield of many plants that are not killed is greatly reduced. Preliminary estimates made by the plant disease survey [4] places the loss in 1917 at 1.5 per cent for the entire country, or about 1,890,000 bushels. The losses are much greater in some sections of the country than in others, being as much as 50 per cent in some fields where especially susceptible varieties are grown. In other sections there are no losses, owing to the fact that only nonsusceptible varieties are grown, or that the disease has not yet been introduced.

The disease is more or less generally distributed and the percentage of loss is greatest in New Jersey, Delaware, Maryland, Ohio, Illinois, Iowa, Kansas, and California. In some other States, however, the disease is somewhat restricted to localized areas where the losses in some instances are quite high. In New Jersey and some other States a reduction of 50 per cent in yield is not uncommon, and 95 per cent of the plants are occasionally diseased. In Ohio and Illinois, where the industry is restricted to a relatively small district, the losses from stem rot vary from 5 to 40 per cent. In Iowa and Kansas losses as high as 60 per cent have been noted. The industry in all of these States just mentioned is confined to isolated sections where the crop has been grown extensively for a number of years and to a variety especially susceptible to the disease.

It is important to note in this connection that in these districts much of the soil is so light and sandy as almost to preclude its use for the cultivation of other crops. It is in just such soils and not the heavy and more fertile ones that stem rot is most destructive.

Symptoms

In the seed bed advanced stages of stem rot are easily detected after one has become familiar with the disease. The foliage is duller in color, yellowed between the veins, and somewhat puckered. (Pl. 1,

[4] United States Department of Agriculture, Bureau of Plant Industry. Estimate of crop losses due to plant diseases, 1917. U. S. Dept. Agr., Bur. Plant Indus. Plant Disease Survey Bul. 2: 14. 1918. [Mimeographed.]

B.) It is not uncommon to see a cluster of plants in the bed showing the characteristic symptoms of the disease. Stem rot in the seed bed usually can be detected in the white part of the stem below the surface of the soil by a faint purplish tint that is cast by the blackened fibrovascular bundles through the epidermis. The diseased fibrovascular bundles can be seen still better by breaking open the cortex.

Advanced stages of stem rot are usually detected in the seed bed and the affected plants discarded. Nevertheless, slight infections may be overlooked when the plants are set out. Such plants soon develop the disease and die early or produce only a few small potatoes. Field infections also take place throughout the growing season, and some of the infected plants may live throughout the summer and produce a normal crop.

The disease symptoms of the leaves of infected plants in the field and in the seed bed are very similar. The youngest leaves generally show signs of the disease first, but usually remain attached till the plant dies. (Pl. 1, B.) On the other hand, the oldest leaves drop off soon after the fungus enters the petioles. When a young plant is attacked, a number of short stems may develop at the center of the hill, producing a rosetted appearance. The internodes of the stem and the petioles of the leaves are sometimes shortened, and the former may be considerably thickened. This condition alone, in the absence of other characteristic symptoms, is sufficient to arouse suspicion of the presence of stem rot. The fibrovascular bundles of the diseased vines may be discolored as far as 3 to 8 feet from the roots. In the later stages the cortex ruptures, the blackened tissue becomes exposed, and typical spores of the fungus are produced thereon. The vines may die after forming a few small potatoes. These potatoes often produce sprouts an inch or more in length, which in turn are killed by the invasion of the fungus. The causal organism grows downward from the stem, producing a black discoloration of the fibrovascular bundles of the roots. (Pl. 2.)

There are no external symptoms that will reveal with certainty the presence of the stem-rot fungus in the stored potatoes. The blackening of the fibrovascular ring in stored potatoes, visible only when the potatoes are cut, is no guaranty of the presence of the stem-rot organism, since the fibrovascular bundles of healthy stored sweet potatoes are frequently darkened. Also, if the vines are frosted in the field or the potatoes badly chilled before they are dug, the fibrovascular bundles may turn dark. Frequently, however, small sprouts one-half inch or more in length grow out from stem-rot infected roots in the fall before they are dug. These sprouts come mostly from the stem end and often form in a little cluster at one or two points on the potato. These sprouts sometimes persist throughout the storage period. It is possible in such cases to pick out stem-rot roots in storage by these external symptoms.

Causal Organisms

Halsted (*62*) was the first to attribute stem rot to a fungus. There is no evidence, however, that he proved by inoculation experiments

that *Nectria ipomoeae* was the true cause of the disease. He was probably led to such a conclusion by the fact that *N. ipomoeae* was frequently found on the decayed portion of stem-rot plants, and for similar reasons he probably concluded a few years later that the ring rot was caused by the same organism (*64*).

In 1911 Harter and Field began a study of stem rot and isolated two species of Fusarium, *F. batatatis* and *F. hyperoxysporum*, with which they produced the disease (*80*). On the other hand, numerous attempts to infect sweet-potato plants with *Nectria ipomoeae* from pure cultures failed. It was thought at first that one of these two species of Fusarium might be the conidial form of *N. ipomoeae*. Halsted showed that it was easy to derive the ascigerous stage of this fungus from the conidia and the conidia from ascospores in pure

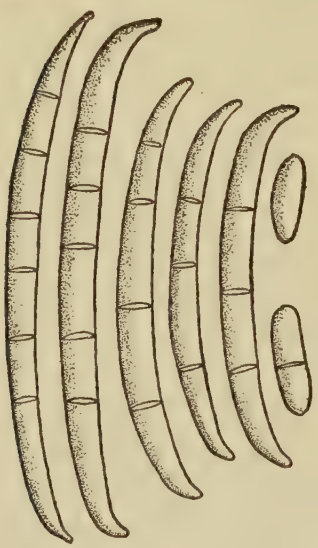


FIG. 2.—Conidia of *Fusarium batatatis*, showing range of size and number of septations. × 1,200

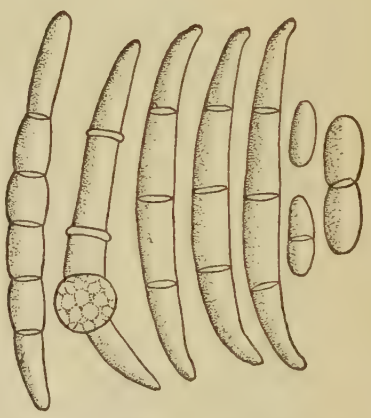


FIG. 3.—Conidia of *Fusarium hyperoxysporum*, showing range of size and number of septations and an intercalary chlamydospore. × 1,200

culture. The writers have obtained similar results, thus confirming Halsted's findings. However, hundreds of cultures of the two species of Fusarium causing stem rot have been grown, and in no case has anything suggesting perithecia ever been produced. The morphological difference between the conidia of *N. ipomoeae* on the one hand and *F. batatatis* and *F. hyperoxysporum* on the other also shows that neither one of the latter two is the conidial stage of *N. ipomoeae*. The conidia of *F. batatatis* (fig. 2) and *F. hyperoxysporum* (fig. 3) are mostly three septate, rarely four septate, while those of *N. ipomoeae* (figs. 4 and 5) are mostly five septate besides being much larger and different in shape. These fungi also differ in their cultural characteristics. Other diagnostic characters have been pointed out by Wollenweber (*210*) in a technical description of the two species.

PATHOGENICITY

That a species of Fusarium and not *Nectria ipomoeae* is the cause of stem rot was first published in 1913 by Harter and Field (*78*). Their initial inoculation experiments were made in 1912 and had for their object the verification of Halsted's results. When it was discovered that *N. ipomoeae* did not cause stem rot, inoculations were made with various species of Fusarium isolated from the fibrovascular bundles of sweet-potato roots and stems. One of these, namely, *F. batatatis*, which was one of the first species isolated, produced the disease. A few months later another species of Fusarium was isolated and its pathogenicity proved. This differed so materially in cultural characteristics from *F. batatatis* (fig. 2) that it was soon suspected of being a different species and was later identified as *F. hyperoxysporum* (fig. 3).

FIG. 4.—Conidia of *Nectria ipomoeae*, showing range of size and number of septations. × 1,200

FIG. 5.—An ascus of *Nectria ipomoeae*. × 1,200.

The initial inoculations with *Fusarium batatatis* and *F. hyperoxysporum*, as well as with *Nectria ipomoeae*, were made with sweet potatoes growing in the greenhouse in pots of soil that had been steamed for one hour at 15 pounds' pressure. Both the former two species produced typical symptoms of stem rot, while the latter did not. Some of the plants inoculated with *N. ipomoeae* died in a manner somewhat resembling "damping off," but the fungus penetrated the fibrovascular bundles only about one-half inch from the wound and did not produce any of the symptoms of stem rot. All these preliminary experiments were made during the winter months or early in the spring when the temperature of the greenhouse was not ideal for the growth of sweet potatoes. Similar experiments were repeated later in the greenhouse and in the field with a considerable number of plants when the temperature was higher.

The organisms recovered from previously inoculated plants of both the parasitic species were inoculated into healthy plants, and the disease was again produced. In the field 200 inoculations were made with *Fusarium batatatis*, and 61.5 per cent of the plants became infected. At the same time 99 and 100 plants, respectively, were inoculated with *F. hyperoxysporum* and *N. ipomoeae*. Of the former 92 per cent but none of the latter became infected.

Other fungi, as, for example, *Gibberella saubinetii* (Mont.) Sacc., *Fusarium oxysporum* Schlecht. (several different isolations), *F. caudatum* Wr., *F. radicicola* Wr., and *F. orthoceras* Ap. and Wr., were isolated from rotted portions of the roots and stems, none of which, when inoculated into plants growing in the greenhouse and in the field, produced any symptoms of stem rot.

All of the inoculations in the above-mentioned experiments were made by inserting hyphae and spores into wounds made in the plants at the surface of the soil, a method that will doubtless give a higher percentage of infection than any other. Experiments more nearly approximating field conditions were performed by mixing pure cultures of *F. batatatis* and *F. hyperoxysporum* into soil previously steamed for one hour at 15 pounds' pressure and setting the plants in it. Plants set in naturally infested soil have become infected (*93*), showing that it is not necessary to insert spores or hyphae into wounds in order to obtain infection, the percentage of infection, however, being much lower than when the plants were wounded. It must not be forgotten that every sweet-potato plant is wounded when pulled from the mother potato, and it is likely that it is through such or similar wounds that most infection takes place under natural conditions, although the hyphae may penetrate the root hairs or epidermal cells directly.

From the point of inoculation the fungus grows down into the roots and out into the vines. The fungus has been inoculated into the vine at the axis of the fourth leaf, about 1 foot from the base of the plant, and infection resulted. Vines have also been found in the field naturally infected at some distance from the hill, usually at the node. From this point it grew in both directions, invading the roots and other vines of the same plant.

As might be expected, young plants are more susceptible to the disease and usually succumb. No real difficulty has been experienced in infecting half-grown plants, although they are apt to survive and produce a partial or full crop of potatoes.

The parasitism of the two stem-rot organisms is thought to be restricted to the sweet potato with one exception to be pointed out later. This question can not be definitely settled, however, since it is impossible to subject all or any great number of species of plants to infection. Plants which it was thought might be naturally susceptible to infection by these fungi were sought for among those closely related botanically to the sweet potato or among those generally grown in rotation with sweet potatoes. The susceptibility of several species of Ipomoea commonly found in sweet-potato fields and consequently subject to natural infection were tested by inserting spores and hyphae into wounds at the lower part of the vine of young plants. The following plants were inoculated: *Ipomoea purpurea* Lam., *I. hederacea* Jacq., *I. coccinea* L., and *I. lacunosa* L. *Ipomoea hederacea* alone became infected when inoculated with both *Fusarium hyperoxysporum* and *F. batatatis*. These species of morning-glory have frequently been examined in sweet-potato fields where stem rot was common, but in no case have any infected plants been found, from which it is concluded that they are not susceptible to the disease under natural conditions.

The following cultivated crops frequently grown in rotation with sweet potatoes have been inoculated with one or the other of the

stem-rot organisms: *Solanum melongena* L., *Capsicum annuum* L., *Trifolium pratense* L., *S. tuberosum* L., and *Lycopersicon esculentum* Mill. These experiments were conducted with plants grown in pots in sterilized soil in the greenhouse during the summer months when the temperature was best suited for infection. None of the plants showed the slightest indication of stem rot, although there was in most cases a blackening of the fibrovascular bundles for a centimeter or so about the wound, from which the fungus could be recovered. These and other inoculation experiments have demonstrated that certain organisms may penetrate the fibrovascular bundles a short distance and cause the formation of necrotic tissue in the vicinity of the wound from which the organism may be recovered without producing symptoms of the disease.

In some sections of Maryland where sweet potatoes and tomatoes are grown on a commercial scale both the stem rot and wilt occur. The theory is frequently advanced that the wilt of tomatoes is more prevalent when tomatoes follow sweet potatoes in the rotation. This suggested the possibility that the two diseases were caused by the same fungus, although this seemed unlikely in view of the characteristic morphological differences between *Fusarium lycopersici* Sacc. on the one hand and *F. hyperoxysporum* and *F. batatatis* on the other. Cross-inoculations were made, using cultures of the organisms isolated from sweet potatoes and tomatoes, each fungus having been previously proved to be parasitic on its respective host. The results of these investigations (*85*) showed that the organisms were pathogenic to the hosts from which they were isolated, but that the sweet-potato fungus was not pathogenic to tomatoes, and vice versa. These experiments were carried out during two summers with the same result.

In 1917, sweet-potato plants affected with stem rot were collected in Orange, Stanislaus, and Merced Counties, Calif., and *Fusarium hyperoxysporum* was isolated from them. This organism was found to be identical with the same species isolated from material in the Eastern, Southern, and Southwestern States. During the summer a number of sweet-potato plants growing on the Potomac Flats, near Washington, D. C., were inoculated with this strain, and the disease was produced in 100 per cent of the plants.

Both *Fusarium batatatis* and *F. hyperoxysporum* are widely distributed, although the latter species alone was isolated from California material. The two species, however, have been isolated from plants grown in many other parts of the country. *F. hyperoxysporum* is the more common of the two, it being obtained in 75 per cent or more of the isolations.

That *Fusarium hyperoxysporum* is more often isolated than *F. batatatis* may not seem so surprising when it is recalled that it is the more vigorous parasite of the two. A résumé of the experiments shows that infections were obtained in 75 to 100 per cent of the inoculations with the former species and on an average very much less frequently with the latter, although in a few cases a high percentage of infection was obtained with *F. batatatis*. Under field conditions the difference is very striking. *F. hyperoxysporum* evidently has the ability to infect to a degree not possessed by *F. batatatis*. Experiments have demonstrated that the percentage of infection by both species is influenced by the age of the plants.

The influence of temperature on infection and on the development of a disease is a subject of considerable importance but one on which no large amount of work has been done. It is well known that some fungi, like *Phytophthora infestans* (Mont.) De Bary, require cool weather for infection. On the other hand, some organisms require a comparatively high temperature. Gilman (*57*) has shown that a relatively high temperature is required for the infection of cabbage by *Fusarium conglutinans* Wr., and Tisdale (*189*) has shown the same for the infection of flax by *F. lini* Bolley. Other investigators have shown that optimum temperature for infection varies with the organism. As regards the two species of Fusarium parasitic to sweet potatoes, all the evidence seems to indicate that relatively high temperatures contribute to infection of the host and development of the disease thereafter. The percentage of infection from inoculations in the greenhouse during the winter months was generally low. The temperatures were relatively low for the greater part of the time, usually not exceeding 75° to 80° F. during the warmest part of the day. On the other hand, when the experiments were conducted during the summer months when the temperature was high but otherwise under similar conditions, the percentage of infections was high, and the symptoms produced on the host were typical of those found under field conditions. It has frequently been observed by the writers and often noted by farmers that stem rot is not conspicuous in the field until the advent of warm weather. The information obtained with respect to the temperature requirements of these organisms soon taught the writers to wait for warm weather before conducting experiments either in the greenhouse or in the field. From the data published and observations made it would not be surprising if species of Fusarium, as a group, should be found to require high temperatures for normal infection and subsequent development. It was also shown, experimentally, that soil temperature and soil humidity play an important rôle in infection (*93*).

The optimum temperature for infection was found to be about 30° C., the maximum 35°, and the minimum as low as the plant will grow. Infection occurred over a wide range of soil moisture. Plants in soil so dry as barely to support plant growth became infected. At 28 and 75 per cent of the water-holding capacity of the soil, 94 and 100 per cent, respectively, of the plants became diseased.

The results of some experiments dealing with the loss of parasitism of the stem-rot fungus in the absence of its host may appropriately be recorded here. *Fusarium hyperoxysporum*, the species employed in many successful inoculation experiments, was first isolated June 20, 1912. During the same year 100 plants growing in the field were inoculated, and 92 per cent became infected. This organism was kept in culture, and inoculations were made with it each year for a period of five years. Before being used for inoculations it was rejuvenated as much as possible by being grown under the best known cultural conditions. In 1913 a number of plants were inoculated and a high percentage of infections obtained, the fungus apparently having lost none of its ability to infect. In 1914 it was less virile, only about 50 per cent of the inoculated plants becoming infected. In 1915 only a very small percentage of infection was obtained, and the symptoms

produced in some of the plants could hardly be classed as typical of stem rot. In 1916 none of the inoculated plants showed the slightest symptoms of the disease. Here seems to be a case where an organism known to be very actively parasitic in the beginning and carried in culture by the method best known to the writers had actually lost the ability to infect its original host. These results might be interpreted to mean that the benefit derived from crop rotation is not through the dying out of the causal organism, but rather to its loss of parasitism in the absence of its normal host. In view of the fact that many fungi can be grown indefinitely in culture as saprophytes, there is no reason to doubt that they might grow saprophytically for many years in cultivated fields. Sherbakoff (*158*) and Link (*113*) also suggested the possibility that species of Fusarium may lose their virulence when grown in culture media. On the other hand, Bisby (*16*) found that *F. oxysporum* after having grown in culture for three years still had the power to infect potato stems and to rot the tubers. Edson and Shapovalov (*45*) found that age did not lessen the pathogenicity of the species of Fusarium studied by them, while Burkholder (*19*), on the other hand, found that, in a period of five years, *F. martii phaseoli* Burk. had changed morphologically and physiologically and in its ability to infect the host.

MORPHOLOGY

While *Fusarium batatatis* and *F. hyperoxysporum* cause identical pathological symptoms, they differ morphologically. The conidia of *F. batatatis* (fig. 2) and *F. hyperoxysporum* (fig. 3) are from 11 to 13 and 8 to 9 times longer than broad, respectively. The apical cell of the former is slender, but that of the latter is bottle shaped. The basal cell of *F. hyperoxysporum* is pronouncedly more pedicellate than that of *F. batatatis*. The one-celled conidia predominate in the latter species, though they are generally intermixed with dorsiventral conidia having three or more septations. Normally *F. batatatis* does not have a pionnotes stage, while a perfect pionnotes is the rule for *F. hyperoxysporum* after one or two transfers on sweet-potato stems or cooked Irish-potato cylinders. *F. batatatis* may be induced to develop a perfect pionnotes by repeatedly isolating and transferring sickle-shaped spores, the tendency, however, being to return to the production of the one-celled spores, which give a typical powdery appearance to the surface of steamed potato cylinders. On sterile sweet-potato stems *F. hyperoxysporum* produces a pionnotes consisting of a slimy mass of dorsiventral spores and a minimum of hyphae. *F. batatatis*, on the other hand, does not produce a pionnotes under similar conditions, but forms a cottony growth of hyphae that produces mostly one-celled conidia. Both species form blue sclerotial bodies in culture on steamed sweet-potato cylinders, those of *F. batatatis* being small and numerous and those of *F. hyperoxysporum* being fewer and larger.

Fusarium hyperoxysporum and *F. batatatis* differ in the size and shape of their spores (figs. 2 and 3), which in the absence of other diagnostic characters are sufficient to differentiate these two species. The spore measurements are as follows:

Fusarium batatatis.—Unicellular conidia, 5 to 12 by 2 to 3.5 μ; three septate, 25 to 45 by 2.75 to 4 μ; four to five septate, 37 to 50 by 3 to 4 μ. Brown chlamydospores varying from 7 to 10 μ.

Fusarium hypcroxysporum.—Unicellular conidia, 5 to 12 by 2 to 3.5 μ; three septate, rarely four and five septate, conidia from sporodochia, 25 to 42 by 3.25 to 4.75 μ. Brown chlamydospores varying from 7 to 10 μ in diameter.

Other differential characters may be found in an article by Wollenweber (*210*), in which is contained a discussion of the morphological and other differences between these two species, as well as between *F. hyperoxysporum* and *F. oxysporum.*

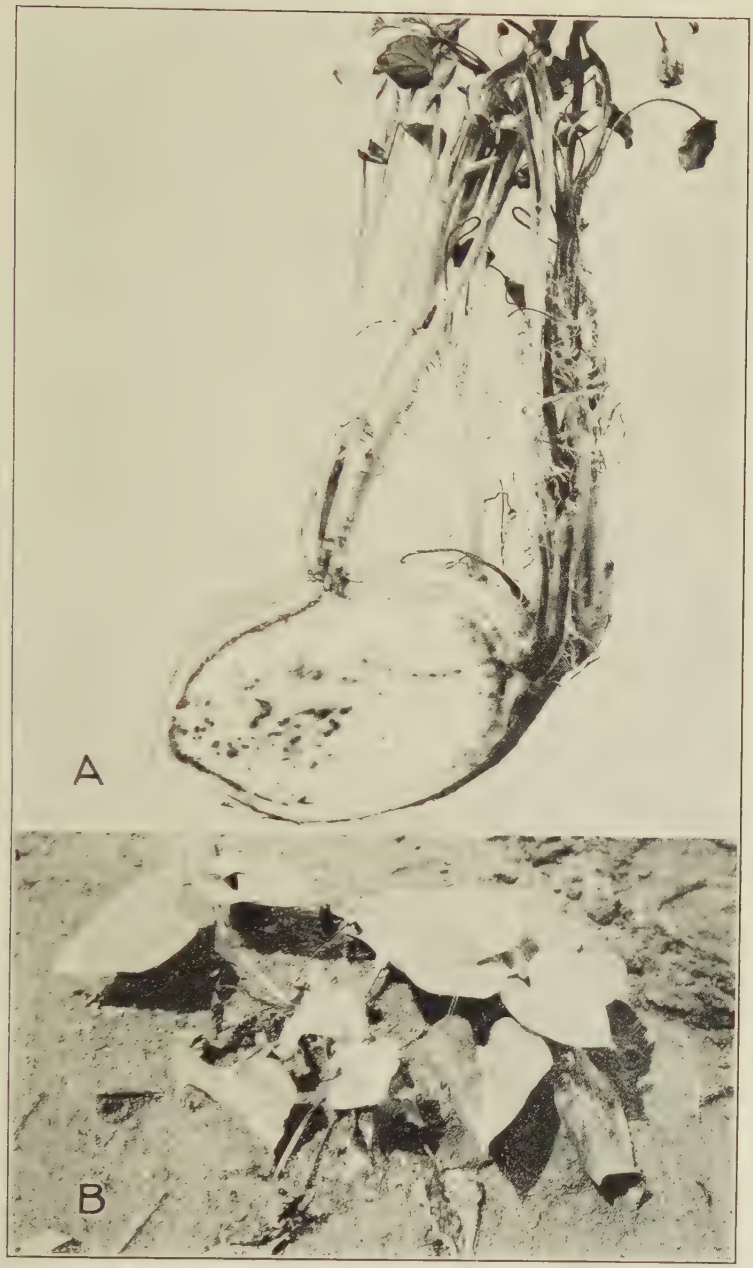
Several species of Fusarium, most of which are vascular parasites belonging to the same general group and possessing many characteristics in common, might be confused with *F. hyperoxysporum.* So far as known, none of them are parasites of the sweet potato. There is then a biological difference, which assists in the separation of these closely related species. The species with which *F. hyperoxysporum* might be confused are *F. vasinfectum* Atk., the cause of cotton wilt; *F. tracheiphilum* (E. F. S.) Wr., the cause of a wilt of a species of Vigna; *F. lycopersici* Sacc., the cause of the tomato wilt; *F. nivale* (Fries) Sor., the supposed cause of a wilt of a species of Citrullus; and *F. oxysporum*, the cause of a wilt of Irish potatoes. Perhaps in most cases a Fusarium isolated from the fibrovascular bundles of the stems or roots of these plants would be tentatively assumed to be the species causing a wilt of that particular crop. Such identifications are not always reliable for permanent records, since cultural and morphological differences must be sought. Such differences between the species mentioned above have been pointed out by Carpenter (*22*).

LIFE HISTORY

The life history of *Fusarium batatatis* and *F. hyperoxysporum* is the same. The conidial stage is the only one known. Chlamydospores are developed in the hyphae and frequently in the conidia themselves. These thick-walled dark bodies serve conveniently to carry the fungus through unfavorable weather conditions. However, with a crop cultivated and handled like the sweet potato such spores are not necessary to the continued existence of the parasite. The stem-rot organisms have been isolated repeatedly from the fibrovascular bundles of stored potatoes at various times throughout the winter. Chlamydospores have never been found in the fibrovascular bundles, and conidia only rarely, and in view of this fact there is reason to believe that the fungus lives over winter in stored potatoes in the mycelial form.

Field experiments have demonstrated that these organisms live over winter in the soil, but in just what form is not known. Conidia are frequently produced in the field on vines killed by the fungus, and it is probable that chlamydospores are formed also on such vines. Both species have been kept in culture for a year or more and readily revived from the spores. They, therefore, like many other fungi, possess considerable power of resistance to drying.

There are two possible sources of infection—(1) the seed bed and (2) the field. If diseased potatoes are used for seed the fungus frequently, though not always, grows from the fibrovascular bundles (pl. 1, A, and fig. 6) of the potato into the slips in which it is carried into the field. Field infections are common. The roots of every plant are wounded when pulled from the mother potato, so that all plants are subject to infection and a considerable percentage of them



STEM ROT (FUSARIUM HYPEROXYSPORUM AND F. BATATATIS).—I

A.—Sweet potato taken from a hotbed and sectioned so as to show the blackened fibro-vascular ring and its connection with the sprouts. The fungus has grown from the potato into the sprouts. B.—A characteristic stem-rot plant in the field. Note the dead leaves lying on the ground, also the yellowed or copper-colored apical leaves on the vines

STEM ROT (FUSARIUM HYPEROXYSPORUM AND F. BATATATIS).—II

A plant obtained from the field rather late in the season and cut open in such a way as to show blackened fibrovascular bundles extending from the stem into the roots. The fungus will grow from the stem into the roots in the field and from the potatoes into the sprouts in the seed bed

actually become infected when planted in infested soil. As high as 60 per cent of perfectly healthy plants have become diseased when grown in soil where the stem-rot fungi were present.

The earliest infections of the season take place either in the seed bed or in the field soon after the plants are set out. The first symptoms, depending somewhat on weather conditions, may appear in about two weeks after planting. Throughout the growing season other plants become diseased, although often at digging time a good crop of potatoes may be harvested, which suggests that infection took place too late in the season to affect materially the productivity of the plant.

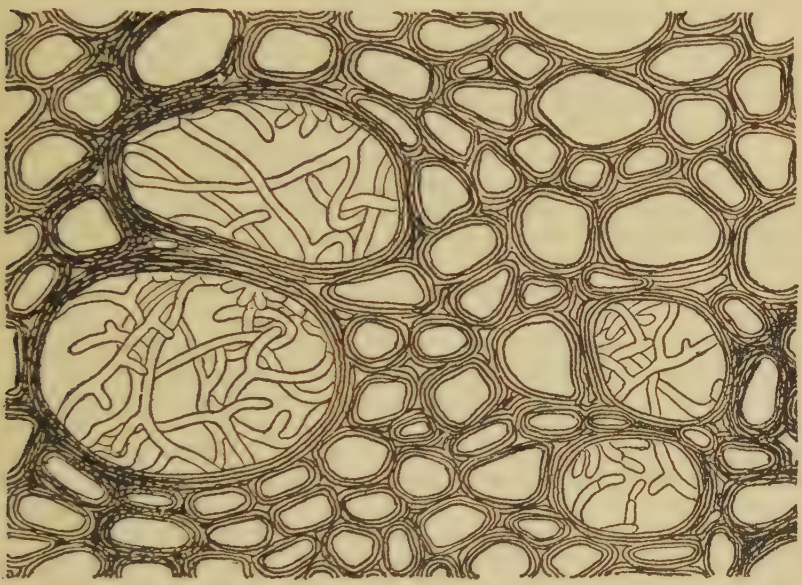


FIG. 6.—Cross section of sweet-potato stem, showing mycelium of *Fusarium batatatis* in the fibrovascular bundle. × 500

DISSEMINATION

The distribution of stem rot, as well as other diseases of the sweet potato, is brought about by the usual channels of trade, in which case the potatoes and young plants are the principal agencies. The dissemination of the stem-rot (or wilt) fungi may appropriately be considered from the standpoint (1) of their distribution over long distances and (2) their distribution over short distances, or from one field or farm to another in the same community.

Seed potatoes as a means of dissemination

From information gathered at certain sweet-potato growing centers, such as may be found in Illinois, Iowa, Kansas, and California, where stem rot is very prevalent, one is led to conclude that its appearance there is the result of the shipment of seed potatoes and slips from the States of New Jersey, Delaware, Maryland, Virginia, and

elsewhere. Importations of seed potatoes in large quantities have been made in the past from these States and are still being made to some extent. It is claimed that the date of the first appearance of stem rot and other sweet-potato diseases in these communities corresponds with the time of the first importation of seed. What almost constituted an epidemic in North Carolina in 1918 resulted from the importation of seed potatoes from another State. The farmers of California have obtained seed potatoes liberally from the East and claim that stem rot and other troubles were introduced with them.

Dissemination by means of plants

If diseased potatoes are used for seed, the pathogen may grow from them into the slips and with them be carried to the field. The percentage of infected slips obtained from diseased potatoes, however, is not as high as might be expected. Out of 23 diseased potatoes bedded in one experiment only 1 produced diseased slips—20 in all. In another experiment 75 selected stem-rot potatoes were bedded. Two months later 320 plants were pulled, and as far as could be judged from a macroscopic examination none had stem rot. Examination 23 days later showed that out of 77 plants 14 had stem rot without question; and 25 days later, of 54 plants examined, 4 had stem rot. It is, of course, probable that many more of these plants would have developed the disease if they had been planted and allowed to grow for a few weeks, although the disease could not be detected macroscopically when the plants were pulled. The shipment of plants has resulted in as wide a distribution of the several diseases as the shipment of potatoes for seed. The growing of plants for sale has become an established industry in many parts of the country. There are at the present time several growers in New Jersey and elsewhere in the northern coastal plains region (and in the South) who ship plants over considerable distances. An examination of the beds revealed the fact that stem rot and black rot were both present. When plants from such beds were examined, a considerable percentage of them were found to be diseased. The yield from field planting of such plants was also greatly reduced. The growing of plants on a commercial scale is even more extensive in Alabama, Florida, Georgia, and other Southern States. In these States thousands of bushels of sweet potatoes are bedded and millions of plants shipped to all parts of the country, even as far west as New Mexico, Arizona, and California. A careful examination of some of these beds and consignments of plants showed that diseased stock was being shipped.

On the other hand, beds were found almost entirely free of both stem rot and black rot, the absence of the diseases being due to the fact that some growers are familiar with the diseases and the methods for their control and make practical application of this knowledge. Moreover, others are indifferent and give little attention to the control or eradication of the diseases and will doubtless continue to do so as long as they are able or are permitted to dispose of their stock.

Local distribution

There are a number of conceivable ways besides the sale or exchange of plants and seed sweet potatoes whereby the several sweet-

potato diseases may be distributed locally, the methods of distribution depending to some extent upon the type of soil, contour of the country, and farm practices.

As many as 25 to 50 per cent of the plants were diseased the first year in fields where sweet potatoes had never been grown. In an experiment designed to test the general distribution of the casual organisms the plants were grown under controlled conditions and were planted on ground on which sweet potatoes had never been grown; yet 40 per cent of the plants developed stem rot. The plants used in the experiment were free from stem rot and black rot, as was shown by the fact that plants from the same bed planted on noninfested soil in another part of the country remained entirely free from the disease. The infestation of the soil in this case may be accounted for by the carrying of the spores from adjoining fields by such agencies as the wind and drainage water. In southern New Jersey and in some other States there are no fields, new or old, that can be assumed a priori to be free from these fungi. High winds prevail in the spring, and clouds of dust are blown about in which the spores are probably borne. Heavy rainstorms are also frequent, so that the spores might readily be washed from one field to another. Irrigation, as practiced in California and some localities in Washington, New Mexico, and Colorado, where sweet potatoes are a minor crop, may conceivably be a means of the distribution of stem rot and other disease-producing fungi. The danger of scattering the spores of various fungi with farm implements and by farm animals roaming from one field to another needs no discussion.

It is the practice among some farmers to feed the decayed or partially rotted sweet potatoes to stock or to throw them on the manure heap to be hauled later to the field. This practice, while followed with the best intentions, illustrates one of the many ways these organisms can be disseminated.

BLACK ROT

History

Black rot, caused by *Ceratostomella fimbriata* (E. and H.) Ell. and sometimes referred to as black shank or black root, is known by several common names, the one used depending either upon the locality where it is found or upon the part of the host affected. Black rot is the name generally applied to the disease of the potato, whereas black shank or black root is often used when referring to decay of the underground part of the stem. The common names used have been established by farmers, who used them to designate the injury to the various parts of the affected plant, not knowing that they had a common cause.

In 1890 Halsted (*61*) published a very clear and accurate description of the disease and of the fungus causing it. One year later (1891) Halsted and Fairchild (*67*) published the results of a thorough and careful investigation of the black-rot organism, and little has been added to our knowledge of the disease or its cause since then. They mentioned the presence of sclerotia, which were suspected of being a fourth type of reproductive body, but which later were shown by Taubenhaus (*176*) not to be in any way connected

with *Ceratostomella fimbriata* but to be a species of Sclerotium. It is evident from a reading of their paper, however, that they entertained some doubt as to the connection of the sclerotial bodies so frequently found associated with the black-rot organism. Halsted (*66*) and Galloway (*56*) later published short accounts of the disease and recommended methods for its control.

Contemporaneously with Halsted, Chester (*26*) carried on experiments looking toward the control of the disease. For 20 years or more after Halsted wrote on this disease very little study was made of it, although reference was made to black rot by Carver (*25*), Townsend (*190*), Wilcox (*207*), Duggar (*42*), and others. The senior writer later worked out effective measures for its control (*70*).

The time and place of the origin of this disease can not be established with any degree of accuracy. Old residents in New Jersey claim to have knowledge of the disease as early as 1868, at which time it was said to be well established and destructive. The writers have shown other species of Ipomoea to be susceptible to artificial infection, but in no case have any of the species been found infected under natural conditions. Species of Ceratostomella are found on other hosts, and it may be that some of them are identical with the species occurring on the sweet potato. If such were the case, a careful study of the facts might lead to a probable answering of the question of origin. Black rot, while reported from foreign countries, is certainly much more destructive and prevalent in the United States than elsewhere, and in view of this fact alone it might be suspected of having its origin here. Such, however, need not necessarily be the case. Sweet potatoes are thought to be native to tropical America, and the disease may have occurred there long before it reached the United States. When brought to a new environment, like asparagus rust when introduced into the United States, it might quickly have become a disease of great destructiveness.

Geographical Distribution and Economic Importance

Halsted and Chester reported black rot from New Jersey and Delaware, respectively, in 1890. In 1892 and 1894 McCarthy (*115*) and Burnette (*20*) reported the occurrence of black rot in North Carolina and in Louisiana, respectively. In 1895 Price (*139*) called attention to the destructiveness of black rot in Texas, and Townsend (*190*) reported it as serious in Maryland in 1899. Barre (*12*) in 1910 mentioned the occurrence of black rot in South Carolina and stated that he was at that time carrying on experiments looking toward the control of this and other storage troubles. In 1903 the disease was reported from Ohio and California and again from Ohio in 1907, and from Indiana, New Jersey, and Tennessee the same year. Burger (*18*) collected black rot in Florida in 1923, and in recent years its occurrence has been reported from various other places in the United States. For a number of year the writers have been collecting data with respect to the distribution of the various sweet-potato diseases, some of which have been obtained by personal visits and some from material sent in for examination from the following States: Virginia, New Jersey, Georgia, Alabama, Florida, Delaware, North Carolina, Maryland, Mississippi, Louisiana, Kentucky, Arkansas, Tennessee, Illinois, California, Iowa, Mis-

souri, Kansas, Texas, South Carolina, Ohio, Indiana, New Mexico, and Washington. Black rot is probably the most widely distributed of any of the field diseases of the sweet potato, being probably coextensive in the United States with the crop itself.

Black rot has been reported from foreign countries as follows: From Haiti (*60*) in 1924; from New Zealand, by Kirk (*105*) in 1907; from Australia in 1908 (*1*); from Hawaii in 1926 (*194*); and from Japan by Ideta (*99*). In October, 1921, the inspection service of the Federal Horticultural Board sent the writers a few partially decayed sweet potatoes obtained from immigrants from the Azores, which upon examination proved to be infected with a fungus identical with the one causing black rot in this country. Specimens of black rot were received through the Federal Horticultural Board from San Juan, Porto Rico, in July, 1924. Although black rot has attracted little or no attention in foreign countries, it is not unlikely that it occurs quite generally throughout the sweet-potato regions. These, however, are the only authentic evidences of black rot in any country outside of the United States of which the writers are aware.

It is not easy to estimate the relative loss to a crop caused by different fungi. It is believed that greater loss is caused to sweet potatoes by the black-rot fungus than by any other organism, with the possible exception of *Rhizopus nigricans*, which, although it may cause a little damage in the field under certain conditions, is primarily a storage-rot organism. Black rot is a seed-bed, field, and storage disease, the combined loss in storage being considerably greater than that in either the field or the seed bed. Individual cases are known where most of the potatoes in storage were rendered worthless by black rot. Other diseases, viz, foot rot, soil rot, and Texas root rot, cause greater loss in isolated sections of the country, but they are for the most part restricted in their distribution. Neal (*126*) states that black rot is by far the most serious disease of the sweet potato in Mississippi, and Barker (*11*) found it causing damage up to 90 per cent of the crop in Haiti. Very little attempt is made to control it there, largely because it is a local crop grown in small plots.

Symptoms

In the early stages of black rot the foliage is yellowed and sickly in appearance, and black cankers (or spots) may develop on the underground parts of the stem (pl. 3, A) contiguous to the potato, caused by the fungus growing from the potato to the stem or at some point between the surface of the soil and the point of attachment to the potato. In severe cases of infection all of the sprouts may be killed before they get through the ground. (Pl. 4, C.) If infection takes place after the plants have reached a considerable size there is little external evidence of the disease, the plants being supported by roots thrown out near the surface of the soil. Farmers usually attempt to discard all plants showing any evidence of the disease, but slight infections may be overlooked, and many infected plants are set in the field. If weather conditions are favorable, most of the plants live and grow, producing a partial or full crop. The part of the stem below the surface of the soil may be rotted away and the plant supported by roots developed from the vines

above the decayed region. Such diseased plants frequently produce a crop. At digging time lesions vary in size from mere specks to spots 1 to 2 inches in diameter. (Pl. 4, A and B.)

Black rot produces on the surface of the potato somewhat circular depressed spots of varying sizes (pl. 5, A and B), depending on the age of the infection. The surface of the spot is of a grayish black color, which turns a dark greenish black when moistened. The fungus usually penetrates only to the fibrovascular ring, although occasionally it extends deeper, as shown by the specimen in Plate 3, B, which was collected in Texas and a pure culture of *Ceratostomella fimbriata* isolated from the innermost part of the decayed tissue.

Causal Organism

Without giving a technical description of the black-rot fungus, Halsted (*61*) created for it a new genus and species, *Ceratocystis fimbriatum* E. and H. Later Saccardo (*149*) technically described the fungus and transferred it to the form genus Sphaeronema, using Halsted's specific name, so that it later was known as *Sphaeronema fimbriatum* (E. and H.) Sacc., the name by which it was known for a number of years.

Elliott (*50*), upon examing material embedded in paraffin, discovered that the fruiting bodies previously known as pycnidia were in reality perithecia and that the small almost spherical spores that are emitted from them were ascospores. There were found to be eight spores in a more or less globose ascus. The ascus wall is very thin and fragile, the spores being released from the ascus before they escape from the perithecium through the long fimbriated beak. Following the discovery of the ascigerous stage of the black-rot fungus, Elliott (*51*) made a cytological study of the processes of fertilization in which he pictured some very curious nuclear phenomena. This organism differs from many other Ascomycetes in that the ascus discharges its spores before it escapes from the perithecium. The spores held together by a mucilaginous substance are emitted in long chains or spore horns through the fimbriated neck of the perithecium, which appears to be only large enough to allow a single spore to pass through it at one time.

Pathogenicity

Ceratostomella fimbriata, which can be obtained readily in culture, seems so obviously a parasite that proof of it hardly appears necessary. Proof that *C. fimbriata* grows from diseased potatoes to the slips produced from them has been obtained by bedding black-rot potatoes in soil previously sterilized by steaming for two hours at a pressure of 15 pounds. In these experiments the potatoes germinated in about 10 days, and in approximately four weeks the fungus had destroyed or invaded most of the slips. As might be expected, some of the plants grew to a fair size, but many of the sprouts were killed before they were through the ground.

The parasitism of the black-rot fungus was further demonstrated by inoculating previously sterilized soil with a pure culture of *C. fimbriata* and bedding therein healthy potatoes disinfected for 10 minutes in a solution of mercuric chloride 1 to 1,000. The potatoes

germinated normally, and in three weeks the characteristic yellowing of the leaves indicative of black rot occurred. At the end of four weeks black-rot cankers were present on the underground portion of the stems, and perithecia had developed in the wounds. At the end of one more week the fungus had attacked the potatoes and perithecia had been formed on them.

Healthy disinfected sweet potatoes germinated in moist chambers have been sprayed with the spores when the sprouts were one-half to 1 inch in length, and at the end of 12 days most of the sprouts and potatoes showed unmistakable evidence of black rot, some of the sprouts already having been killed.

Unsprouted disinfected potatoes have been subjected to infection in a moist chamber by spraying with spores suspended in water, and at the end of 11 days many black-rot spots one-fourth to one-half inch in diameter were present. Inoculations have been made by wounding in various ways. When the potatoes were cut with a knife and spores and hyphae smeared on the wounded surfaces the fungus spread rapidly over the entire cut surface if sufficient moisture was supplied. In one such experiment, after inoculation, the potatoes were wrapped in moistened filter paper and then confined in a damp chamber. At the end of six days the entire cut surface was covered with hyphae and perithecia, the fungus having penetrated a short distance into the unwounded tissue. The growth of the fungus on a cut surface is greatly retarded by the absence of an adequate supply of moisture. Similar experiments in which spores and hyphae were inserted beneath the epidermis gave 100 per cent infection, the lesions attaining a diameter of about 1 inch in four to six weeks. While wounding is not necessary, it contributes to infection (*92*). It has often been observed that the fungus rapidly overruns wounds made by growth cracking in the field or by rodents or other means. When potatoes in the moist chamber were sprayed with a spore suspension the infection generally took place through dead rootlets, and the fungus later grew into the tissue of the potato. On the other hand, when the fungus was mixed with sterilized soil in which healthy disinfected potatoes were bedded the sprouts were first attacked. It is possible that such infections were through wounds, but it is believed that it is not always or necessarily the case. The infection experiments were made with potatoes of all sizes, and no difference in the degree of susceptibility was found.

The control of this, as well as other diseases, depends in part at least upon the susceptibility of other hosts. The plants besides sweet potatoes selected by the writers for inoculation experiments were those commonly found in the sweet-potato fields and closely related to it botanically, with one exception, namely, *Solanum melongena*. Inoculations were made by inserting spores and hyphae into wounds of *Ipomoea purpurea*, *I. lacunosa*, *I. hederacea*, *I. coccinea*, and *I. bona nox* L. growing in sterilized soil in the greenhouse. None of the species of Ipomoea were resistant, there being 100 per cent infection in all except *I. bona nox*, of which only 90 per cent became diseased. None of the plants of *S. melongena* showed any symptoms of black rot. The lesions were in every particular typical of those occurring on the sweet potato, and perithecia were produced in great abundance. Taubenhaus and Manns (*185*) record the occurrence of *Cera-*

tostomella fimbriata on *I. purpurea* and *I. pandurata*, showing that other hosts may perpetuate the disease. The surest evidence, however, that they will do so is the finding of infected plants under natural or field conditions. The writers have examined many plants of the species mentioned above, growing in fields where black rot was abundant, but none have ever been found infected. It is likely that they may be infected artificially and yet not be economically important as hosts for the black-rot organism.

MORPHOLOGY

There is nothing particularly characteristic about the hyphae of the black-rot organism. They are much branched, septate, and hyaline at first, becoming brown with age.

Ceratostomella fimbriata produces two types of conidia: Hyaline conidia (pl. 6, A), and olive-brown conidia. (Pl. 6, B.) The former are produced in chains in great numbers, both on the host and in culture. They are easily broken apart by any slight disturbance to the substratum on which they grow. They germinate readily (pl. 6, F) and under favorable conditions produce another crop of conidia in less than 24 hours. Olive-brown conidia may be produced (pl. 6, I) in the germination of these conidia. The hyaline conidia are one-celled, cylindrical, club-shaped to dumb-bell shaped. They are usually straight, rarely curved, and angular or rounded at the ends. They are extremely variable in length, ranging from 9.3 to 50.6 by 2.8 to 5.2 μ (26 measurements from an 11-day-old culture on corn meal). The conidiophores are hyaline and vary from 50 to 100 μ in length and from 4 to 6 μ in diameter.

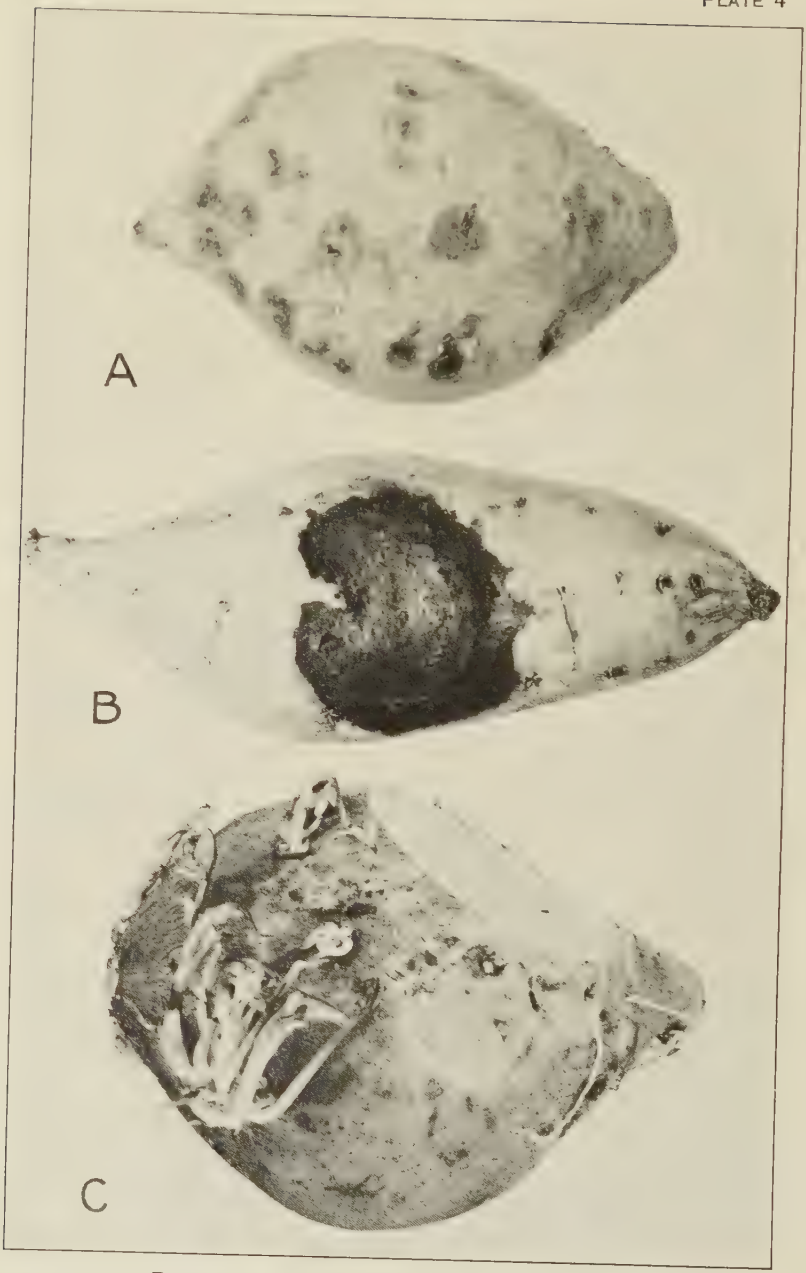
The method of formation of the hyaline conidia has been a subject of recent study by Lehman (*112*), who concludes that the first two spores are formed as endoconidia; that is, instead of the conidia being abstricted from the end of the conidiophore, which is the rule with many such fungi, the first two are formed with a sheath at the end of the conidiophore. After the first two endoconidia are produced the cell wall of the free end of the conidiophore is dissolved, and the conidia are pushed out. Many other conidia are produced within this sheath and are pushed out of the casing by the formation of conidia behind them, none of the conidia except the first two being regarded as true endoconidia.

The olive-brown conidia are formed on rather short conidiophores, either singly or in chains of two to six. They are at first nearly hyaline and thin walled, but soon turn brown and become thick walled with age. They vary considerably in shape, some being spherical, others club-shaped, oval, pear shaped, and ellipsoidal. (Pl. 6, B.) They range in size from 10.3 to 18.9 by 7.6 to 10.3 μ (22 measurements from a 3-month-old culture on corn meal). The brown conidia are produced in much smaller numbers than the hyaline ones and much more abundantly on some kinds of culture media than on others. These conidia are found buried in the diseased tissues of the roots of sweet potatoes nearly to the fibrovascular ring. They germinate (pl. 6, G) by the production of one germ tube.

A second type of reproduction consists in the formation of perithecia (pl. 6, D) within which is inclosed a large number of more

BLACK ROT (CERATOSTOMELLA FIMBRIATA).—I

A.—Two sweet-potato plants pulled from a commercial seed bed, the one at the left showing that infection had taken place by the growth of the parasite from the potato to the stem; the other (right) is from the soil at a point some distance above the potato. B.—Cross section through an infected potato, showing the depth to which the organism will sometimes penetrate. A pure culture was obtained from the innermost part of the infected tissue

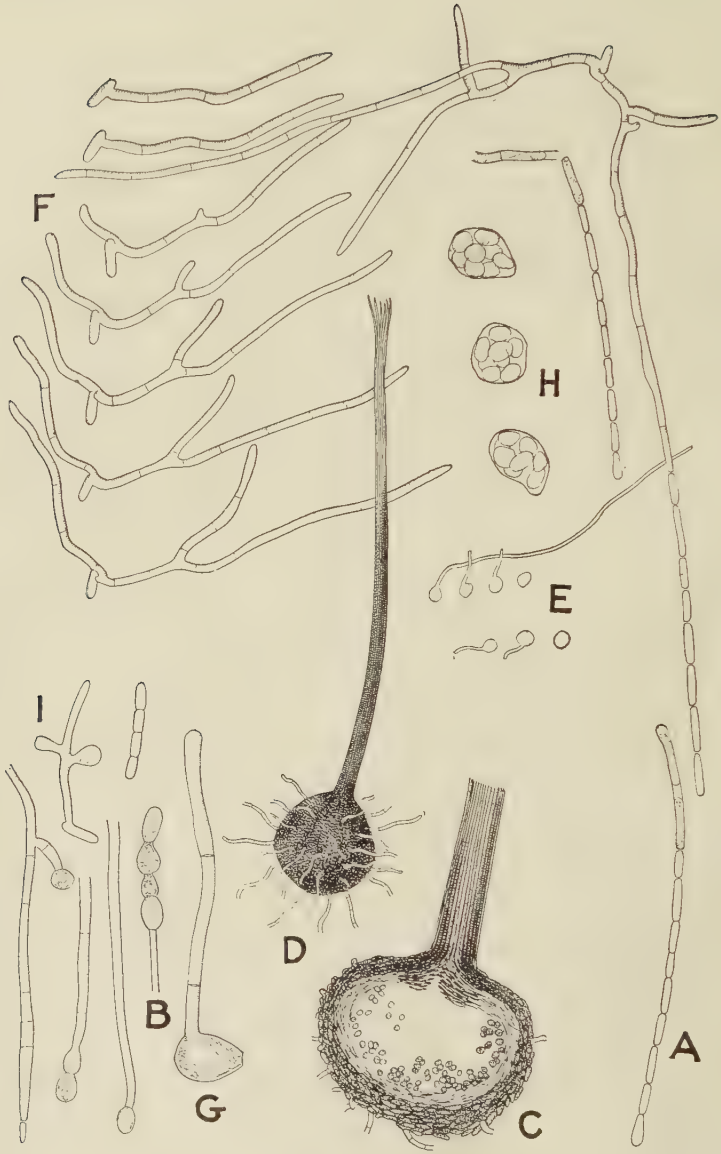


BLACK ROT (CERATOSTOMELLA FIMBRIATA).—II

A.—An artificially infected potato which shows that infection takes place through old dead rootlets or through lenticels or other openings, such as wounds. B.—A potato from the field at digging time, showing the slightly sunken spot with a somewhat circular outline, characteristic of the disease. C.—Infection of young sprouts. These are sometimes killed before they emerge

BLACK ROT (CERATOSTOMELLA FIMBRIATA).—III

A.—A sweet potato obtained from a storage house in the early fall, showing four black-rot spots.
B.—The same potato held for two months at a temperature of from 10° to 13° C., showing to what extent the spots have enlarged



CERATOSTOMELLA FIMBRIATA

A.—Chains of hyaline conidia, × 230. B.—Brown, thick-walled conidia, × 230. C.—Section of perithecium showing asci and ascospores free in the central cavity, × 160. D.—Mature perithecium showing fimbriated beak, × 72. E.—Germinating ascospores, × 340. F.—Germinating hyaline conidia, × 230. G.—Germinating brown spore, × 640. H.—Asci with ascospores, × 640. I.—Brown conidium formed in germination of a hyaline conidium, × 230

or less pear-shaped to spherical asci (pl. 6, H), each containing eight nearly spherical hyaline spores. The perithecia are produced in considerable numbers on the host under natural conditions and in many kinds of artificial culture media. They are in general flask shaped (pl. 6, C and D), with a long, cylindrical beak, and range in size from 105 to 140 μ in diameter. The beak is fimbriated at the apex (pl. 6, D) and varies in length from 350 to 800 μ and from 20 to 30 μ in diameter (12 measurements from 15-day-old cultures on stems of *Melilotus alba*). It is usually straight, but is sometimes slightly bent. The perithecium is composed of a thin, dark outer wall several cells in thickness and a thinner hyaline inner wall. (Pl. 6, C.) The ascospores are 4.8 to 6.9 μ (26 measurements from 11-day-old cultures on corn meal) in diameter and are produced in great numbers. As a result of pressure developed by the absorption of water, they are forced through the long beak in a creamy mass in the fimbriations at the top. Under the microscope they may be seen exuding from the beak in a long spore horn or coil. They are held together in a gelatinous or mucous substance, from which they are released with considerable difficulty, or only after being immersed in water for a considerable length of time. The spores swell in water and germinate by the production of a single germ tube. (Pl. 6, E.)

PATHOLOGICAL HISTOLOGY

An examination of a number of stained sections cut through black-rot lesions shows that the invaded cells are so disorganized as to lose practically all semblance of form. The cells several layers below the surface are invaded by the hyphae and the olive-brown conidia formed within them. These conidia are formed more abundantly on the surface on short conidiophores and frequently in chains. The cells, evidently dead, are badly ruptured, and the protoplasm is abnormal in appearance or, in some cases, entirely wanting. The hyphae ramify in and among the cells. Except in rare cases, the hyphae of *Ceratostomella fimbriata* penetrate only a short distance below the surface. One example has already been mentioned and is illustrated by Plate 3, B. Just below the cells that have been killed by the invasion of the hyphae a cork cambium from four to six cells in depth is laid down. The formation of a cork cambium beneath wounds made either mechanically or by the invasion of fungi is a rather common phenomenon. Weimer and Harter (*200*) found that under suitable environmental conditions wounded sweet potatoes laid down a cork cambium beneath the wound, which became evident at the end of two days and quite pronounced in three. They also found that this cork cambium layer offered some resistance to the decay of sweet potatoes by *Rhizopus tritici*. Tisdale (*188*) likewise found that flax attacked by the fungus *Fusarium lini* formed a conspicuous cork cambium and observed that the fungous hyphae advanced to but never penetrated it. This phenomenon suggested to him the possibility of its connection in some way with the resistance of some of the plants to the wilt. Weimer and Harter observed also that the starch in the region of the cork cambium formed beneath artificial wounds almost entirely disappeared. A similar condition was observed with respect to the starch in the parenchyma cells on either side of the cork cambium

just below the tissue invaded by the black-rot organism. Whether or not the formation of a cork cambium explains why the black-rot fungus does not usually penetrate the host tissue deeply can not be answered definitely at this time. It might be assumed, however, that such was the case and that in those few instances where the fungus was isolated from the deep-lying tissue it had either succeeded in penetrating the cork cambium layer or that conditions were such that no cork layer had been formed.

LIFE HISTORY

Sweet potatoes affected with black rot are generally discarded when dug. It is doubtful, however, if all those bearing visible spots could be discarded, and, even if they could, many infections too small to be seen with the unaided eye would find their way into storage. Under storage conditions these spots enlarge, sometimes involving the entire potato. At the center of the spot a cluster of perithecia is often formed, from which ooze great numbers of spores. These spores are carried about by insects, distributed by the settling of the sweet potatoes in the bins or by workmen preparing the potatoes for market, and scattered by others means, so that, in houses where the disease is present, a general infection of the crop frequently results.

Experiments to prove that black rot is distributed and continues to develop in the storage house have been made by mixing an equal number of healthy potatoes and black-rot potatoes and storing them in bushel containers. When the potatoes were removed at the end of the storage period all but one were affected with black rot.

In the spring of 1913, a number of healthy sweet potatoes were bedded in black-rot infested soil, the plants produced therefrom being planted on soil that was noninfested. When the crop (about 16 bushels) was harvested in the fall, about 1 peck with noticeable black-rot lesions was discarded. The remainder was stored in the usual way in ⅝-bushel baskets. When removed from storage the following spring, 13 baskets of potatoes were affected with black-rot and the causal fungus had spread from this lot of potatoes to those in adjoining bins. The conclusions to be drawn from the above experiment are (1) that the fungus lives over in the soil; (2) that there are infections at digging time too small to be seen with the unaided eye, since all potatoes were carefully examined for black rot before they were placed in storage; and (3) that the fungus spreads in the storage house.

It is likely that the hyaline conidia and ascospores are chiefly instrumental in the rapid propagation and spread of the fungus, both types of spores, especially the latter, being produced in considerable abundance in storage. However, the thick-walled olive-brown conidia are better adapted to carry the fungus through unfavorable environmental conditions. The fungus survives the winter on or in the potatoes in storage and on dead débris in the field. In view of the present methods of handling the crop, any one of the known types of spores might, and probably does, serve the purpose of perpetuating the fungus. Sweet potatoes affected with black rot bedded in sterilized soil have produced infected plants. The fungus has been isolated in pure culture from potatoes carried through the winter in

storage, and experiments have demonstrated that the causal organism lives from one season to the next in the field. Experiments have shown that plants from healthy disinfected sweet potatoes bedded in infested soil became diseased; also that many healthy plants will become diseased if set in a field where black rot was prevalent the previous year.

From these data it is evident that infection may take place either in the seed bed or in the field, or both. In the seed bed the infection of the young plants may result either by the growth of the fungus from the diseased potato to the plant or by direct infection from the soil or by both. If diseased plants are set in the field they may die early in the season, and healthy plants may become infected if planted in infested soil.

DISSEMINATION

The dissemination of *Ceratostomella fimbriata* is brought about in the same way as stem rot, i. e., by the shipment of plants and potatoes to various parts of the country. This subject has been more fully considered on page 17, to which the reader is referred. Inasmuch as the black-rot organism causes a storage disease, it is necessary to mention briefly the distribution of the fungus in the storage house. Under average storage conditions the perithecia are produced in considerable numbers, and spores are exuded from them in creamy masses at the end of the beak. Doubtless the spores are scattered about by air currents, by means of insects which are frequently found in storage houses, by the settling of the potatoes in the bins, and as a result of the handling of the potatoes in preparing them for the market.

FOOT ROT

History

In 1912 Harter discovered foot rot caused by *Plenodomus destruens* Harter, then a new disease, causing heavy losses to sweet potatoes in Virginia. The following year a brief article (*69*) was published in which the characteristic symptoms of the disease as well as the causal organism were discussed. The name "foot rot" was applied to this disease, since the foot or base was the part of the plant most commonly attacked. This name has since come into general use among both scientists and farmers, although the disease is still known to the farmers in some localities in Virginia and other States as "die off."

Nothing is definitely known about the origin of foot rot. Inquiry among farmers in Virginia and other States has failed to give any definite information from which conclusions may be drawn. It seems likely from the evidence obtained that foot rot and black rot were not distinguished, and in view of that fact the former disease may have been prevalent for many years previous to its discovery without having attracted attention.

Geographical Distribution and Economic Importance

In 1913 foot rot was reported by Harter (*68*) from Virginia; in 1915 from Missouri, Iowa, and Ohio (*71*), and later from Maryland, Kansas, New Jersey, California, South Carolina, and Georgia. The

writers collected a number of specimens of the disease in Maryland in 1917, and specimens were sent to them from the same State in 1918 and from South Carolina in 1922. A few diseased specimens were collected in New Jersey in 1917. In 1917 foot rot was found to be very prevalent in California and was reported to occur in the States of Tennessee and Florida, showing that it is widely distributed. So far as its known distribution is concerned, the disease is confined to the northern range of the sweet-potato belt with the exceptions of Tennessee, South Carolina, and Florida. It is uncertain whether this is to be interpreted to mean that the climate farther south is unfavorable to the fungus or whether it has not yet been introduced. If foot rot originated in the northern range of the sweet-potato belt, as seems likely, its introduction into the South would be less likely, since seed potatoes and plants have not until recently been sent south in any great quantities. On the other hand, seed potatoes from Maryland and Virginia have been sent to Ohio, Kansas, Iowa, California, and other Western States. In fact, it has been and still is a common practice to import seed potatoes into these States, and the growers insist that foot rot and other diseases of the sweet potato were not known until seed had been imported from the East.

The total loss from foot rot is not large. Until remedial measures were applied foot rot was very destructive in Virginia, as high as 95 per cent of the plants in some fields being infected. The farmers were on the verge of giving up the industry, but in none of the other States has the loss been so heavy. In Missouri the loss is negligible, there being only a few isolated cases in one or two localities. A survey of the fields about Marietta, Ohio, showed that as high as 75 per cent of the plants in a field of General Grant Vineless were diseased. The sweet-potato industry is small in the State, however, so the total loss from this and other causes is relatively small. The conditions in Iowa are about the same as in Ohio. Foot rot is prevalent, and the percentage of loss is high in some fields. However, stem rot and black rot are very destructive, and complications of the three diseases are such as to render diagnosis exceedingly difficult. In 1917 foot rot was discovered in a few localities in the San Joaquin Valley, Calif., but the loss in no case exceeded 10 per cent. It was observed at Merced, Calif., in 1924. A few years later, however, the disease could not be found in the former region, which indicated that it was probably imported with the seed but was not able to survive the climate there and disappeared with succeeding plantings.

Symptoms

Foot rot in the seed bed causes a stunting and sickly growth or, where the disease is very severe, the death of the plants. The easiest way to distinguish between this disease and black rot, or even stem rot, is to pull up the plants and examine them for the fruiting bodies, which are developed in considerable numbers at and a little above and below the surface of the soil. When the plants are pulled up it is easy to distinguish foot rot from stem rot, since the causal organism of the latter blackens the fibrovascular bundles of the stem. In the early stages black rot and foot rot have rather well-marked distinguishing characteristics. The cankers caused by black rot are more localized and more nearly black than those caused by foot

rot. In the advanced stage of black rot the entire plant is involved, resembling closely the injury caused by foot rot. The pathological symptoms caused by black-rot and foot-rot organisms often merge, so that sure diagnosis is only possible when the fruiting bodies are present.

Most of the initial infections of foot rot take place in the seed bed, the causal organism being carried to the field with the plant, where it continues to develop. The fungus grows slowly, especially in the early stages of infection, requiring three to four weeks, depending somewhat upon weather conditions, to involve any considerable portion of the stem. As soon, however, as the fungus becomes well established it develops very rapidly, and in a week or 10 days completely girdles the stem at 2 to 5 inches (pls. 7, B, and 8, A) above the surface of the soil, killing the plant by the destruction of the cortex. In another week the plants wilt (pl. 8, A) and may die soon thereafter. There is considerable variation in the length of time the infected plants live. Some die rather early in the season, while others linger along to midsummer or even till digging time. In most cases, however, the stem is nearly or completely rotted off, the plant being supported by roots thrown out above the diseased area or possibly by the roots at the nodes of the vines. Pycnidia (pl. 7, B) may be found in considerable abundance in the dead tissues. The growth of the plant is retarded from the beginning of the infection. The foliage turns yellow and some of the older leaves may drop off. These symptoms may be followed later by the wilting of the vines and finally by the death of the plant.

The infection is restricted largely to the stem of the plant at the soil line, although occasionally vines several feet from the stem may be infected at the nodes, the fungus growing in both directions from the point of infection. (Pl. 8, C.) Observations indicate that infections of this type occur only where the vines are protected by a rank growth of foliage.

Not all the plants are killed by the foot-rot organism before sweet potatoes are produced. Many plants live through the entire summer and produce a fair yield. When potatoes are produced on diseased plants they often are infected by the fungus growing on the roots, so that the latter may be rotted at the attached end. These roots, if only slightly infected, are too often placed in storage and serve to carry the disease through the winter.

Field infection of the potatoes is usually at the attached end, the fungus growing from the stem into the roots, and it is not uncommon to find the potatoes in all stages of decay when they are dug in the fall. Those badly rotted are generally discarded, but those only slightly decayed frequently find their way into the storage house, where the disease continues to develop. The loss caused by foot rot in storage is relatively small. The fungus does not destroy the potatoes to the same extent as soft rot or black rot. The causal organism grows slowly, forming a dark-brown firm rot (pl. 8, B) with the development of pycnidia at the surface (pl. 7, A). It often invades wounds, bruises, and tissue killed by other fungi and grows from such spots to the slips in the hotbed. In view of this fact, care should be taken when selecting seed to discard potatoes that are bruised or have other surface spots.

Other fungi cause a rot which in some stages is very similar to that produced by the foot-rot organism. In such cases the only sure proof of the cause of the decay is the isolation of the fungus from recently rotted tissue.

Causal Organism

IDENTITY

The organism causing foot rot was described by Harter (*69*) as a new species of Plenodomus, *P. destruens*. The fungus does not fit well into the genus but is better there than in any of the closely related genera, such as Phoma, Phyllosticta, and Phomopsis. It falls naturally into the order Sphaeropsidales and is similar to the above genera, especially the form genus Phomopsis, but differs from it in essential details.

The diagnosis of the form genus Plenodomus as found in Saccardo's Sylloge Fungorum (*149*) is somewhat brief. In 1911 Diedicke (*36*) worked over this genus and indicated characteristics that separate it from Phomopsis, the genus with which it is likely to be confused. The studies and conclusions of Diedicke were largely followed in determining the genus to which the foot-rot organism belongs. It apparently was not identical with any of the species hitherto described and was accordingly described as new.

Another disease of sweet potatoes, dry rot, caused by *Diaporthe batatatis* Harter and Field, has for its conidial stage a Phomopsis. Dry rot is primarily a storage rot, although it does sometimes occur in the field, causing a decay of the stem and developing fruiting bodies on the stem and vines. The root rot, on the other hand, is primarily a field disease, but it does occur to some extent in storage. Under field conditions it is sometimes difficult, if not impossible, to distinguish dry rot from foot rot macroscopically. With the aid of the microscope and by means of cultures the two are found to be very different. They also differ culturally and parasitically. Foot rot causes large losses in the field by destroying the stem; dry rot very slight losses.

PATHOGENICITY

The pathogenicity of *Plenodomus destruens* has been established by a large number of inoculation experiments of different types. In 1912 and 1913 sweet-potato plants (81 in all, grown in the greenhouse in pots of sterilized soil) were inoculated at the base of the stems by inserting spores and hyphae into wounds. Of the inoculated plants 91 per cent developed the disease, while the 57 control plants remained healthy.

Plenodomus destruens has never been found attacking the foliage, and attempts to infect the leaves artificially by spraying with spores suspended in water were unsuccessful.

Although wounding may be an aid to infection, it is not necessary, as is shown by the inoculation of a number of plants by smearing the pycnospores on the stem just above the ground. The controls rubbed by a scalpel remained sound, but 70 per cent of the inoculated plants became infected. In another experiment spores in water were poured around the plants growing in sterilized soil, and 70 per cent

finally developed the disease. The controls about which water alone had been poured remained healthy.

It has already been pointed out that infections of the vines sometimes take place several feet from the hill. (Pl. 8, C.) In one experiment 100 per cent infection was obtained where inoculations were made at the nodes several feet from the hill.

While the results of the inoculations in the greenhouse seemed to prove conclusively the parasitism of the organism, there is always the possibility that results so obtained might not be the same under field conditions. With this possibility in mind, inoculation experiments were conducted in the field, with the result that all the inoculated plants developed the disease. If there was any difference in the results, it was that the plants inoculated in the field succumbed more readily than those inoculated in the greenhouse.

Plants of three wild species of Ipomoea commonly found in sweet-potato fields, viz, *I. hederacea*, *I. purpurea*, and *I. coccinea*, have been subjected to infection. All of the plants of *I. hederacea* and *I. coccinea* succumbed to the disease, and the causal fungus was recovered from each. While these species may be considered as a possible host for the fungus, infected plants have never been found under natural conditions.

All the above-described inoculations were made with an organism isolated from a diseased sweet-potato plant obtained from Virginia in 1912 or with this organism after it had been recovered from plants inoculated with it. During the summer of 1917 a number of thrifty sweet-potato plants growing on the Potomac Flats near Washington, D. C., were inoculated with *Plenodomus destruens* isolated from plants grown in California, and 90 per cent became infected.

The inoculation experiments described above were made on the Yellow Jersey and Big-Stem Jersey varieties. Although the disease has been observed on a few other varieties in the field, such as the General Grant Vineless and Southern Queen, there was no evidence at hand as to the relative susceptibility or resistance of other commercial varieties. In 1917, 12 well-known commercial varieties were grown in pots of sterilized soil in the greenhouse and inoculated with an organism already shown to be pathogenic to the Yellow Jersey. The varieties were Yellow Jersey, Pierson, Big-Stem Jersey, Yellow Strasburg, Red Jersey, Red Bermuda, Southern Queen, Dooley, Yellow Yam, Pumpkin Yam, Vineless Pumpkin Yam, and Triumph. None of these varieties exhibited any marked degree of resistance to foot rot. From 60 to 100 per cent of the plants were infected, with the exception of the Vineless Pumpkin Yam, of which only 25 per cent became diseased. Judging from the results of these experiments, there is little hope that the disease can be combated by the introduction into infested areas of any of the common commercial varieties.

Although found in storage, *Plenodomus destruens* can not be regarded as a storage-rot organism in the same sense as *Ceratostomella fimbriata* and *Rhizopus nigricans*. That the organism does cause decay is evident from the fact that it has often been isolated in pure culture from sweet potatoes rotted in storage and that the typical rot has been produced by inoculation in the laboratory.

MORPHOLOGY

The mycelium of *Plenodomus destruens* differs very little from that of the other species of the genus. It is much branched in culture and for the most part hyaline, although it produces some brown hyphae in old cultures. Oil droplets or globules are present in the mycelium at all ages and on almost any media. Intercellular, rarely terminal hyaline spherical and oval, thick-walled bodies, 8 to 13 μ in diameter, borne singly or in chains, occur in most substrata at all ages of growth. Brownish bodies morphologically similar to the hyaline ones, but occurring mostly at the end of the hyphae, are frequently found in old cultures.

The pycnidia are buried at first, but later break through the epidermis and appear as black dots scattered over the surface. They stand close together (pl. 7, A and B) on the stem and roots, but they are rarely if ever confluent. They are irregular in form and vary greatly in size, averaging about 300 μ through their greatest diameter.

The outer wall of the pycnidia is dark to almost black. The structure of the pycnidia on the stem appears to differ from that on the root, where there is a well-defined hyaline layer almost equal to the outer wall in thickness. The dark outer wall of the pycnidia on the stem is more conspicuous and better developed than on the roots, the inner hyaline layer being nearly or completely lacking. The sporophores, which are from 6 to 13 μ in length and very narrow, are short, fragile, somewhat inconspicuous, and arise from the inner hyaline layer.

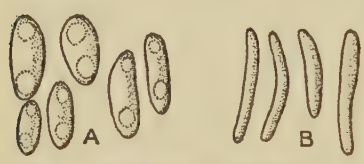


FIG. 7.—*Plenodomus destruens:* A, Conidia developed in artificial media; B, stylopores taken from the host. × 1,200

The spores are discharged through a beak of varying length, which may arise from any part of the upper surface of the pycnidium. In old dried specimens the upper portion of the pycnidium may fall away.

The hyaline, one-celled, and sometimes slightly curved conidia (fig. 7, A), are oblong, rounded at both ends, 6.8 to 10.0 μ long by 3.4 to 4.1 μ wide, with two large oil droplets.

Besides the pycnospores, somewhat cylindrical hyaline curved or straight bodies varying in length from 5 to 15 μ with rounded or tapering ends are sometimes found in the pycnidia from the host and occasionally in those on rice and on sweet-potato stems. (Fig. 7, B.) Several attempts to germinate these bodies were unsuccessful. Their function is not known, but it is probable that they are homologous to the stylospores of the genus Phomopsis (*35*).

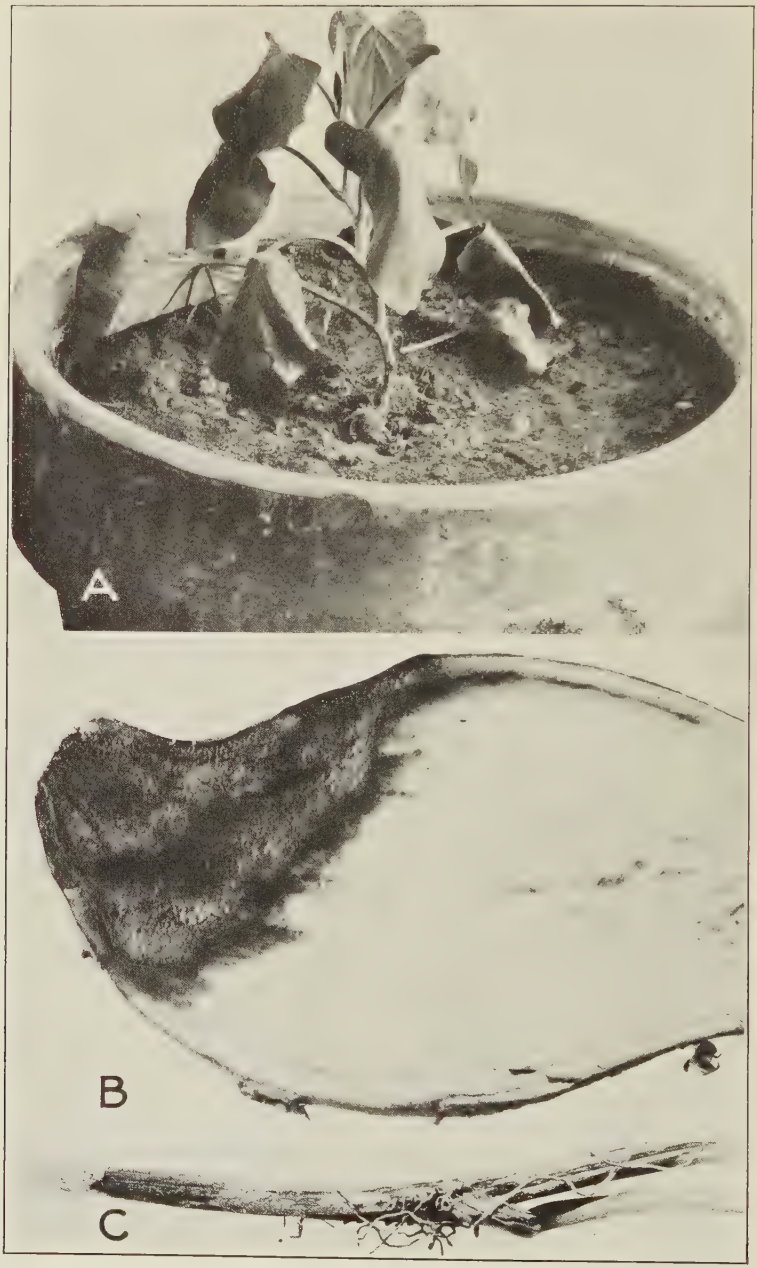
LIFE HISTORY

No ascigerous stage of *Plenodomus destruens* is known. Therefore the only known means of reproduction is by pycnospores, which are produced in great numbers. A few field infections have been definitely proved to occur, but usually the disease is carried to the field on the plants from the seed bed.

Foot rot, like several of the other sweet-potato diseases, is carried through the winter on the potatoes in storage and to a much less

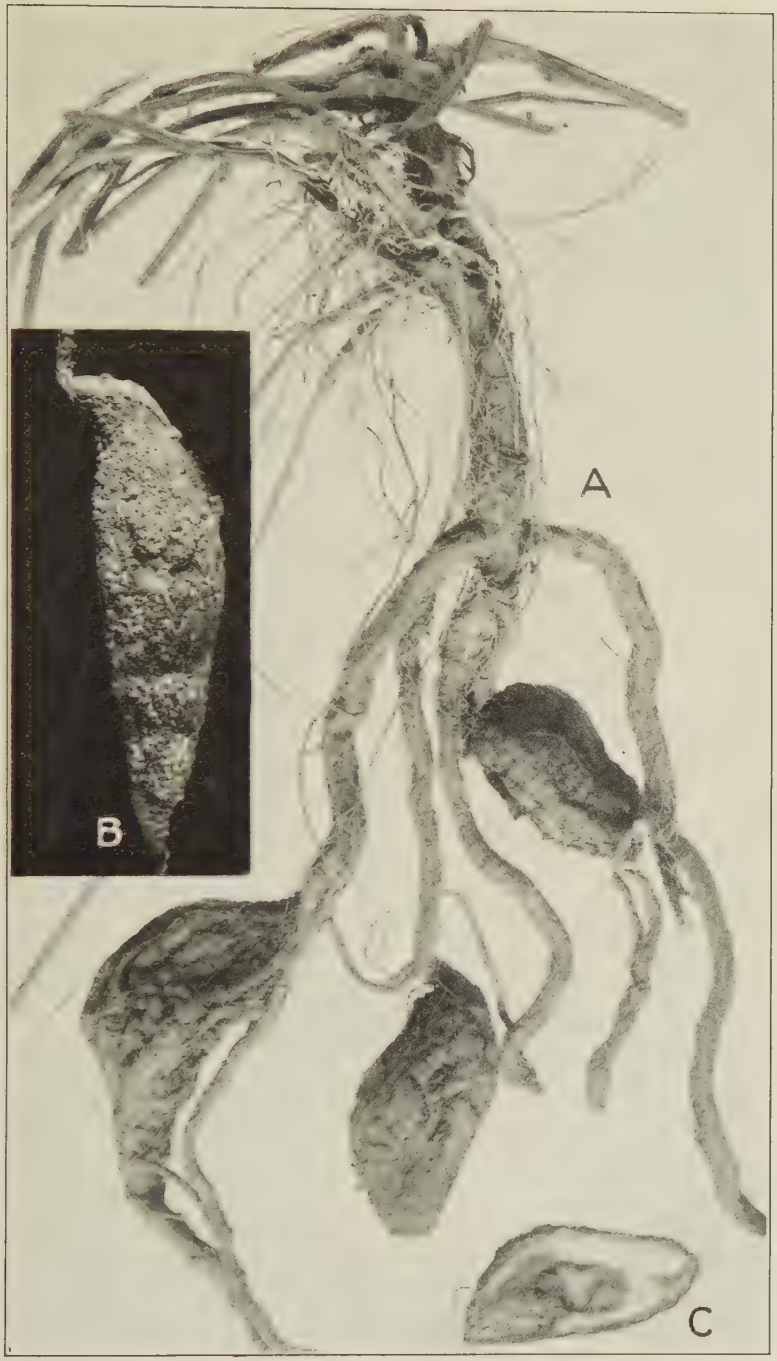
FOOT ROT (PLENODOMUS DESTRUENS).—I

A.—Root after some months in storage, considerably enlarged to show the presence of the pycnidia. B.—The lower part of the stem of a plant which has been rotted off about 1 inch below the surface of the soil. The dead tissue extends about 5 inches up on the stem. Pycnidia are present in abundance on the dead parts of the stem



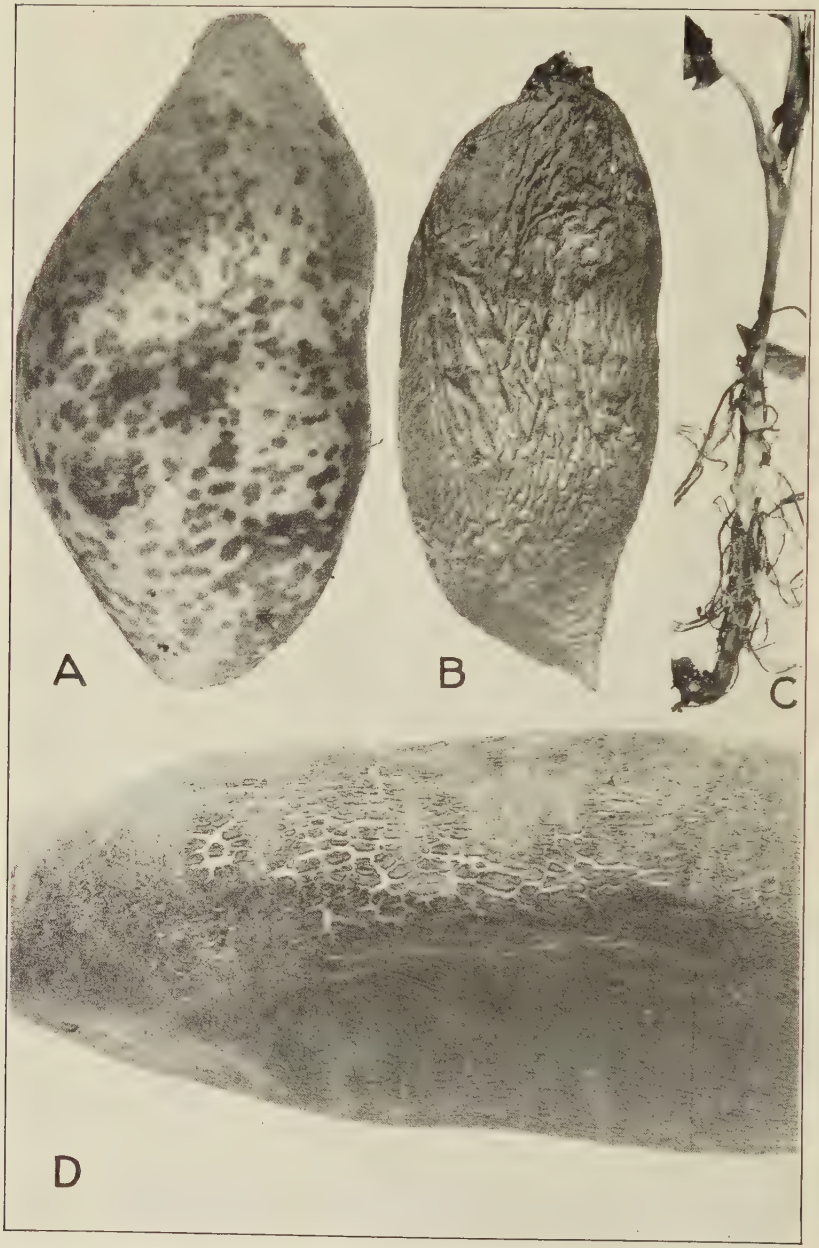
FOOT ROT (PLENODOMUS DESTRUENS).—II

A.—The wilting stage of a plant artificially inoculated with a pure culture of the organism. The stem just above the surface of the soil is partially decayed. B.—A potato partially decayed as a result of artificial inoculation. C.—A field infection of a vine at the node. The organism has extended some distance in each direction from the node. A pure culture of the organism was obtained from this specimen



TEXAS ROOT ROT (PHYMATOTRICHUM OMNIVORUM)

A.—Whole plant affected with root rot, showing mummified roots. B.—Mummified root, showing mycelial masses. C.—Longitudinal section of a root, showing extent of rot



SCURF (MONILOCHAETES INFUSCANS)

A.—Scattered spots resulting from artificial inoculation. B.—A badly shrunken potato from storage, with scurf over the entire surface. C.—Scurf on the underground portion of the stem. Infection probably came from the potato. D.—Cracking of the surface of a sweet potato, caused by the scurf organism

extent in the soil in the seed bed and in the field. The small number of infections of healthy plants set in the field where foot rot was prevalent the previous year shows that the principal source of infection of the plants is in the seed bed. In localities where the disease is prevalent the organism can be found in potatoes in the storage house and in the banks, where a slow, firm, brown decay is produced. The mycelium grows throughout the potato, but the pycnidia are formed only at the surface. During the storage period fruiting bodies may be found in wounds made in the fall at digging time, which, in view of the fact that often only a slight decay or none developed, indicates that the spores were carried to these wounds some time after the beginning of the storage period. In fact, it is reasonable to assume that the spores are scattered promiscuously throughout the house during the storage period. This assumption is strengthened by the observation that foot rot often develops in wounds after the potatoes that showed no evidence of being diseased have been held for a few days under warm conditions. If such potatoes are used for seed, the spores germinate in the seed bed and infect the young plants. These infected plants may be set in the field and may produce some potatoes, which in turn are liable to be infected. In other words, the causal organism is carried with the infected potatoes to the seed bed, from the seed bed to the field with the plants, and thence in the fall to the storage house in or on the potatoes, thus completing a circuit that assures its perpetuation.

TEXAS ROOT ROT

History

Texas root rot, caused by *Phymatotrichum omnivorum* (Shear) Duggar (*Ozonium omnivorum* Shear), is perhaps the disease best known, since it affects also cotton and alfalfa. Various theories were advanced as to its nature before Pammel (*129*) found it to be caused by a fungus. According to some investigators it was caused by an excessive accumulation of alkali, but others attributed it to an excess or deficiency of other soil substances. It was generally agreed by all to be worse following some crops and more or less restricted to certain types of soil.

No real advance was made respecting the cause and nature of Texas root rot until Pammel began a study of the disease. As a result of investigations in Texas for two summers, he concluded that it was caused by a sterile fungus, which not only attacked cotton but many other crops as well. The sweet potato was one of the plants specifically mentioned by him as one of the hosts. He tentatively attributed the cause of the disease to *Ozonium auriconium* Lk., but he was skeptical concerning the correctness of the identification, because it did not agree perfectly with the description of that species.

The work of Pammel was followed by similar studies in Texas in 1901 and 1902 by Duggar (*39*) and in 1907 by Shear and Miles (*157*) and more recently in Texas and Arizona by Peltier (*132*). A number of new hosts were added, and it was pointed out that the grasses and cereals were immune to the disease—a fact of considerable economic importance when a crop rotation is being planned for its

control. It is now known to attack a large number of plants widely separated in relationship, among them being many plants of economic importance as well as weeds.

Geographical Distribution and Economic Importance

Texas root rot, so far as known, occurs only in Texas, Oklahoma, New Mexico, California, and Arizona. Although it may be restricted to spots of various sizes in the field, as pointed out by Pammel, Duggar, Shear, and Miles, such is not always the case. These infested spots, which enlarge from year to year, can be easily detected in the case of cotton and some other crops. It is very destructive to the sweet-potato crops in some parts of Texas and New Mexico, especially in the black, poorly drained soils, where, according to Taubenhaus, it frequently prevents the farmers from growing the crop. The infected areas are not so readily recognized, since in many cases the vines cover the ground, even though the roots may be attacked and the potatoes partly or completely destroyed. The disease may become so destructive that not more than 10 per cent of a crop may be produced. It is much less destructive to sweet potatoes in Oklahoma and has never been reported on this crop in Arizona. In the Pecos Valley of New Mexico considerable loss to sweet potatoes and other crops has resulted from Texas root rot. It is also reported from Mexico by Ramírez (*140*).

Symptoms

On the symptoms on sweet potatoes Pammel (*129*) says:

> In the Texas disease the fungus begins to work on the surface, but in the advanced stages the whole root is found to be covered with the brown threads of the fungus. In some specimens sent to me, and considered sound, I found a number of depressions, in the center of each a small whitish protuberance composed of a mass of fungous threads similar to those on the roots of cotton. The cells adjoining the depression were soft and undergoing decomposition. The rot may begin at the end and work gradually over the entire root. The vines grow on vigorously and show no external symptoms, but at the time of maturity nothing else but the decayed remains of potatoes can be seen, while on the borders of the patch diseased potatoes of all sizes showing different stages of the fungus can be found.

To the above description may be added the fact that the fungus, besides destroying the roots, invades the vines for 6 to 12 inches from the soil line. It may enter the potato at the end or form lesions of varying sizes on the surface, in either case causing a firm, dry rot. Whether this dry rot is caused by the Ozonium alone or as a result of secondary organisms that follow it is not certain. The potatoes finally become hard and mummified, and brownish to buff in color (pl. 9, A, B, and C); on their surface are formed coarse strands composed of a number of brown hyphae, which run more or less parallel to one another. Aboveground the fungous growth is confined within the stem and may be detected by the brown discoloration produced.

Causal Organism

Identity

The disease was attributed by Pammel to *Ozonium auriconium* Lk., although he recognized that the Texas root-rot fungus differed

essentially from that species. The organism was known by that name until Shear (*155*), after having examined Link's type material, described it as a new species, *O. omnivorum* Shear. The organism was studied again in 1916 by Duggar (*41*), who found in Texas and Arizona cotton fields in which there was an abundance of one-celled hyaline spores associated with the characteristic hyphae of *O. omnivorum*, and later by King (*103*). Duggar succeeded in germinating some of the spores, which resulted in the production of hyphae identical with the hyphae previously recognized as those of *O. omnivorum*. On the strength of his results he placed the fungus in the genus Phymatotrichum, the organism becoming accordingly *P. omnivorum* (Shear) Duggar. Duggar admits that it does not fit perfectly in the above-named genus, or, as a matter of fact, in any established genus of the Hyphomycetes, but it is better there than in any of the related genera.

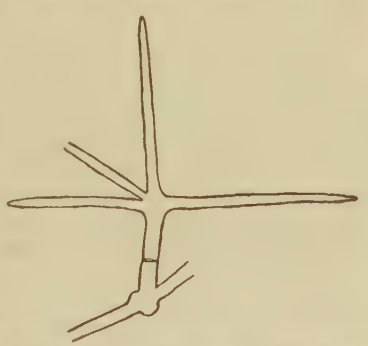


FIG. 8.—Acicular mycelium of *Phymatotrichum omnivorum*. × 750

All inoculation experiments made by Duggar with the fungus from the conidia gave negative results. In view of this fact, it may be questioned whether or not the conidia found by him belong to *Ozonium omnivorum*. Shear (*156*) in 1903 found a Hymenomycete on the stem of an Osage orange (*Maclura aurantiaca* Nutt.) in Texas in close association with typical mycelium of the Texas root-rot fungus, and on the strength of this association described the organism as *Hydnum omnivorum*. He was unable, however, to demonstrate the connection between the Ozonium and the Hydnum, since the latter fungus failed to grow in culture.

PATHOGENICITY

Pammel, Duggar, and Shear and Miles have studied extensively the root-rot disease in Texas, but none of them have recorded successful inoculation experiments. It appears that *Ozonium omnivorum* has been determined by them as the cause of the trouble wholly on the basis of its constant association with the disease.

MORPHOLOGY

According to Duggar, there are three fairly distinct types of mycelium, i. e., the large-celled type, the strand hyphae, and the acicular type. (Fig. 8.) The large-celled type, measuring as much as 20 μ in diameter with cross walls 60 to 120 μ apart, is strikingly similar to Rhizoctonia and is often found abundantly on the margins of the conidial areas. With respect to the strand hyphae, Duggar (*41*) says:

> In these the individuality of the hyphae is practically lost, the strands being ultimately plectenchymatic bands in which the individual cells vary considerably in diameter, the larger cells of young strands resembling somewhat the larger hyphae above mentioned. It is interesting to observe that they may serve not only to spread the fungus vegetatively, but superficial soil strands may function as a conidial stroma. They are also more or less sclerotial and are doubtless an important factor in the persistence of the fungus in the soil.

The acicular type derived from the arachnoid mycelium is fairly rigid hyphae branching in pairs, opposite, and at right angles. (Fig. 8.) The branching is sometimes also verticillate. The branches are rigid, needlelike, and taper to a fine filament 3 to 5 μ in diameter. This type of mycelium has been found only on the roots. There are, of course, intermediate types between the forms here mentioned.

In the early stages of growth the hyphae are nearly hyaline, but they become brownish with age. According to Shear, the sterile mycelium is a dirty-yellow color, sometimes whitish when young, both in culture and in the fibrovascular bundles of the plants.

Duggar, the first to find conidia associated with the Texas root-rot fungus, found that the spores were produced at first on the characteristic larger hyphae and on small branches of those hyphae that make up the strands of the fungus in the soil. The attached conidia were found in heads about short swollen but not necessarily spherical branches of the short-celled or strand hyphae. These branches were simple or forked, the forking being at irregular intervals, and occasionally branching was continued from a swollen cell. The fertile hyphae arise irregularly from the large-celled mycelium or directly from cells of the strands. They are simple or forked with the spore-bearing portion vesicular, often 20 to 28 μ in length and 15 to 20 μ in diameter. The spores are hyaline, spheroidal to ovoidal, the spheroidal averaging 4.8 to 5.5 μ in diameter, the ovoidal 5 to 6 by 6 to 8 μ, extreme diameters 3.2 and 9.8 μ. The conidial stage forms a continuous pulverulent, sometimes crustlike area on the soil.

LIFE HISTORY

Texas root rot does not appear on the plants in the seed bed, being destructive only in the field. It has been reported by Pammel as being evident in the plants in the field as early as May 6. It is not, however, till some time later than this, usually from about the middle of June to July 1, that it becomes very noticeable. In August it is probably most conspicuous and destructive.

If Duggar's work is accepted, the root-rot fungus has a single fruiting stage, namely, conidia, which are borne in great abundance probably only late in the summer. It is very doubtful, however, whether conidia are necessary to the perpetuation of the organism. It is likely that in a climate as mild as that of Texas the mycelium lives through the winter; in fact, it is probable that in some parts of Texas where winter crops are grown that the root-rot fungus continues its growth throughout the entire year.

SCURF

History

The scurf disease of sweet potatoes was first recognized by Halsted (*61*), who published a brief account of it in 1890. To the fungus he gave the name *Monilochaetes infuscans* Hals., a new genus and species, of which, unfortunately, he gave no technical description. This disease was not studied again until the writers and others about 22 years later took up an investigation of it, together with other

sweet-potato diseases. From these studies a better knowledge of the causal fungus, together with methods for its control, has been obtained.

Geographical Distribution and Economic Importance

Scurf is as widely distributed in the United States as the sweet-potato crop itself. It occurs practically everywhere and on practically all varieties, though on some varieties more commonly than on others. Typical specimens of scurf-infected potatoes have been collected in California and New Mexico and from all the Southern States, as well as from New Jersey, Delaware, Maryland, Illinois, Iowa, and Kansas. Outside of the United States the only authentic report of its occurrence is in the Hawaiian Islands, where it is said by Carpenter (*23*) to be present. It probably occurs in Japan (*99*) also.

There are no varieties, so far as known, entirely immune to scurf.

As compared with the loss from black rot, that from scurf in the United States is relatively small. It is uncertain to what extent the yield of the crop is reduced by scurf, the loss being largely restricted to the reduction in the market value caused by the discoloration of the skin and to the loss from shrinkage in storage of badly infected roots, many of which must be thrown away. The potatoes are for the most part infected in the field before the crop is harvested, although the infection centers may enlarge slightly in storage, and possibly a few new ones may be formed. It is not unlikely that there is some increase in the disease under storage conditions, if the relative humidity of the storage house is high. Scurfy potatoes sell for 25 to 50 per cent less than clean ones, depending naturally on the degree of infection.

Except in severe cases of infection, the food value of the potato is not impaired by scurf. The injury to the potato is superficial; the hyphae in penetrating the epidermal cells cause injury, which results in the loss of water. In potatoes slightly infected the loss of water is very gradual and slow, and shrinkage is correspondingly slight, but the loss from badly infected roots (pl. 10, B) is rapid, so that after a period of one or two months in storage an appreciable percentage is unfit for market. Naturally the shrinkage is greatest in storage houses where the temperature is high and the relative humidity low.

Symptoms

Scurf is characterized by a brownish discoloration of the epidermis of the underground parts of the plant. These discolored areas may be in spots (pl. 10, A) or they may cover a considerable portion of the ends or other parts of the potato, large spots often being formed by the coalescing of two or more small ones.

Monilochaetes infuscans is another of the sweet-potato fungi that spread from the infected seed potatoes to the slips and are carried on them to the field. (Pl. 10, C.) The disease is less prevalent in light, sandy soils and worst in heavy soils and in those containing a considerable quantity of organic matter. It may be observed in the fall at harvest time that the greatest number of infected spots

occur at the upper end of the potato, suggesting that the spores have been washed down by the rain, starting new infections, or that the mycelium spreads to the roots from the slips.

Although field infections do occur, the evidence seems to indicate that infected potatoes used for seed purposes are the primary sources of the disease. Sweet potatoes badly affected with scurf are dark brown to nearly black in color and are frequently covered with small, fine furrows or cracks. (Pl. 10, D.) Under such conditions moisture escapes from the potato, which becomes tough, leathery, and event-usually much shrunken and wrinkled after a short period in storage. (Pl. 10, B.)

Causal Organism

IDENTITY

Halsted considered the scurf fungus as belonging to a new genus and species, to which he gave the name *Monilochaetes infuscans*. A detailed study of the causal organism was afterwards made by Harter (*72*), who technically described both the genus and species.

PATHOGENICITY

The pathogenicity of *Monilochaetes infuscans* was proved in the following manner: Sound potatoes were thoroughly washed in water and placed in moist chambers with moistened filter paper in the bottom. They were then sprayed with a suspension of spores and bits of hyphae in sterile water and exposed to the conditions of the laboratory. Sterile water was added from time to time in order to maintain a high humidity in the damp chambers. At the end of two weeks infection (pl. 10, A) centers appeared indiscriminately over the entire surface of the potatoes, these centers gradually enlarging either by the merging of two or more spots or by the enlargement of a single lesion. In artificial inoculations the spots, however, appear not to enlarge by the branching and creeping of the hyphae over the surface but rather by the dropping of the spores, which are borne on conidiophores standing erect or at an acute angle to the surface of the potato, from which new infections start outside the point of the original growth.

MORPHOLOGY

The mycelium of *Monilochaetes infuscans* is hyaline at first, but later turns brown, the terminal cell of the conidiophore remaining hyaline. The vegetative growth on the host and in artificial media differs considerably in general appearance. On the former it is scarcely, or not at all, branched and appears to be reduced to merely a septate conidiophore. The fungus penetrates the epidermis and enters the tissues of the host only a few cells deep. The conidiophores, for such they appear to be, arise from the surface of the host and are attached to it by an enlarged end cell (fig. 9, B) beneath the epidermis. Occasionally two or three enlargements or bulblike cells are formed in a chain, consisting of the portion of the fungus in the host tissue. The conidiophores are unbranched, septate, vary in length from 40 to 175 μ, and bear at the end one-celled spores (fig. 9, A), which are hyaline to slightly brown with age. The conidia

are 12 to 20 μ in length by 4 to 7 μ in width and are formed singly at the end of the conidiophore. As soon as one conidium is mature it separates easily from the conidiophore, and another is formed by the enlargement of the end cell of the conidiophore, to be dropped in turn when mature.

Monilochaetes infuscans is slow growing both on the host and in culture. Some difficulty therefore was experienced at first in obtaining it in pure culture. After a number of trials it was found to make a slow growth on Irish potato, string-bean, and oatmeal agars. By thoroughly washing the potatoes and then disinfecting them for about one minute in bichloride of mercury (1 to 1,000) and planting bits of tissue in one of the agars mentioned above (oatmeal agar is the best) a pure culture could generally be obtained. In a week to 10 days transfers were made to tubes of rice cooked in water or to moistened corn meal, on both of which a matted growth of dark-brown hyphae developed by the end of three to four weeks. Spores are abundantly produced in tubes of cooked rice. Growth has been observed on a few of the common culture media, namely, potato agar, beef agar, rice agar, oatmeal agar, string-bean agar, potato cylinders, sweet-potato stems, and stems of *Melilotus alba*. The general appearance of the growth is not characteristically different on any of these media, forming in all cases a feltlike mat 2 to 3 millimeters in height with an entire margin. The mycelium in mass is almost charcoal black. Following the production of the hyaline conidia, which are formed more abundantly on some media than on others, particularly on oatmeal agar, beef agar, and string-bean agar, a grayish color is developed on the surface. At room temperature (23° to 26° C.) growth is visible in 4 days on all media used, except on rice agar and on stems of sweet potatoes and *M. alba*. In 13 days a small growth developed on rice agar, but on stems of sweet potatoes and sweet clover there was no evidence of growth at the end of four weeks.

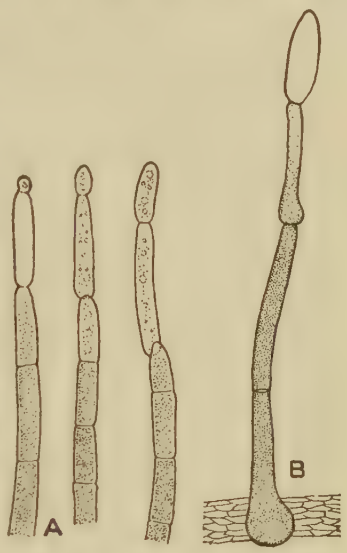


FIG. 9.—*Monilochaetes infuscans*. A, Conidiophores and conidia (× 400); B, cross section through sweet-potato epidermis, showing bulbous basal cell of conidiophore (× 335)

Judging from a limited number of experiments, the fungus does not seem to have a very wide range of temperature for growth. At the end of 24 days only a sparse growth occurred on string-bean agar, rice agar, and oatmeal agar at temperatures ranging from 6° to 7° C. and only a slight growth on oatmeal agar in 14 days at temperatures varying from 30° to 32°.

The conidia germinate slowly on rice or in sweet-potato decoction by the production of one or two germ tubes, usually from the end of the spore, which in 24 hours attain a length about equal to the

length of the spore. The branching of the hyphae begins in about two days and the production of the brown pigment in about three days.

LIFE HISTORY

Only the conidial stage of *Monilochaetes infuscans* is known. The conidia are very sparingly produced, or, at least, are difficult to find under natural conditions. A few spores similar to those of *M. infuscans* have been found on scurfy potatoes, but in no instance were they attached to sporophores, so that it is a question whether they were not spores of another fungus. In moist chambers, however, they are sparingly produced. It is doubtful whether the spores are necessary or even serve in any degree to perpetuate the fungus. The hyphae, like those of other fungi causing sweet-potato diseases, doubtless live over on the stored potatoes and on dead débris in the fields and in the soil. The fungus grows from the scurfy seed potato to the sprouts, on which it is carried to the field and from which it passes to the roots.

That the scurf organism lives over from one season to the next in the soil in the field and in the old seed beds, as well as on sweet potatoes in storage, has been repeatedly demonstrated by field and laboratory experiments. In most of these experiments vine cuttings were set in pots and the pots stationed in such a way that the new vine growth was suspended in the air. By this method it was reasonably certain that the vines were free from the spores and the mycelium of the scurf organism. These vines when about 3 feet long were cut into sections and planted in the field in soil that grew scurfy sweet potatoes the previous year. At the end of the season scurf was found on all of the plants. Similar cuttings in the same soil, which had been steamed as a control, grew clean potatoes. These experiments were duplicated the following year with similar results. Vine cuttings were set in a field where sweet potatoes have never been grown, and a clean crop was produced, which indicates that *Monilochaetes infuscans* is not present in all soils. Plants from scurfy potatoes were also planted in the noninfested soil, and each produced scurfy potatoes, demonstrating that the disease is carried from the potatoes with the plants to the field.

SOIL ROT

History

The soil rot of sweet potatoes, first discovered by Halstead (*61*) in New Jersey, was attributed by him to a fungus to which he gave the name *Acrocystis batatae* E. and Hals. Although he evidently studied the disease with considerable care, giving a fairly accurate picture of its occurrence and symptoms, apparently he did not succeed in isolating the causal organism or in successfully infecting healthy plants. He pointed out that the disease was very destructive in some fields, causing an almost total loss in some cases. For several years following his first description of the soil rot, Halsted gave further attention to a study of the causal organism and methods for its control. In the publication referred to above, he gave several text figures of what he assumed to be the causal organism, although he gave no evidence of having isolated it. His conclusions as to the cause of the

disease were apparently based upon the constant association of a certain fungus with soil-rot lesions. None of his published data prove that he successfully worked out control measures, although he demonstrated that the use of flowers of sulphur (*66*) applied in different quantities per acre reduced to some extent the percentage of soil-rot infected potatoes; also that the total yield increased with the increase in the proportion of sulphur applied.

Following Halsted's investigations, no work on soil rot was undertaken until 1914, when Taubenhaus (*178*) undertook some preliminary studies, in which he points out certain characteristic symptoms of the disease. With respect to its control he found that lime increased the amount of the disease, whereas acid phosphate decreased it.

In 1916 Elliott (*46*, *47*) published two papers, in one of which he set forth the claim that soil rot is caused by a slime mold, and in the other he described in detail the morphology and life history of the organism. Two years later these articles were followed with one by Taubenhaus (*180*), which apparently had for its primary object a confirmation of the work of Elliott. This latter paper, however, contains considerable additional data. Taubenhaus claims to have shown that the organism causes a similar disease of potatoes and turnips and intimates that the tomato and beet may also be susceptible. He suggests that Morse and Shapovalov (*125*) and Ramsey (*141*), who attributed the "pit" disease of the potato to *Rhizoctonia* sp., were in reality dealing with the sweet-potato pox organism, the injury by *Rhizoctonia* sp. being merely secondary. So readily did Elliott and Taubenhaus isolate the slime mold and inoculate sweet potatoes with it that the former recommended it as one suitable for use in the classroom.

The name "soil rot"—frequently referred to by farmers as ground rot, bugsting, pit, pox, and wormhole—was given to the disease originally by Halsted. Taubenhaus later proposed the adoption of pit or pox as a common name for this disease. The writers, while admitting that either pit or pox describes the disease in its late stages better than soil rot, prefer to maintain the old established name by which it was first known in the literature.

Geographical Distribution and Economic Importance

It is not certain that soil rot occurs outside the United States, although there are reasons to believe that it is present in Japan, since, in a publication in the Japanese language (*100*), a disease of sweet potatoes due to *Acrocystis batatae* is mentioned. In the United States it is fairly widely distributed, having been collected, received, or reported from New Jersey, Delaware, Maryland, Virginia, North Carolina, South Carolina (*12*), Oklahoma (*111*), Texas, Florida (*18*), Kansas (*174*), Mississippi, and Arkansas. It is probable that the disease occurs in some other States.

The losses from soil rot vary greatly in different localities. As early as 1890 Halsted found the disease in some fields causing nearly a total loss. It is equally bad in Delaware, and Taubenhaus (*183*) claims that soil rot is one of the worst diseases in Texas, ranking next to black rot in economic importance. Notwithstanding the fact that soil rot has been known a long time, it has not become so widely distributed as some of the other diseases; neither has the loss

been so extensive, except in a few isolated cases. The comparatively small loss caused by it may be explained by the fact that it is very little or not at all carried by the seed potatoes, its distribution being accomplished only by the transfer of soil from the field.

The report of the plant-disease survey for 1919 [5] gives losses for several of the States as ranging from 26,000 to 143,000 bushels. The greatest loss occurred in Texas. Virginia reported a loss of 96,000 bushels and Maryland and Delaware 45,000 and 29,000 bushels, respectively.

Symptoms

The symptoms of soil rot differ markedly from those of any of the other sweet-potato diseases. In extreme cases of infection the plants are dwarfed, often failing to produce more than one or two short vines during the entire growing season. The plants in general appear as though conditions were very unfavorable for their growth, the leaves being small, thin, and pale green in color. Some of the plants may even die before the end of the season. The root system is poorly developed, and in fields where the disease is severe no roots or only a few small ones are produced. On lateral feeding roots, which are usually few in number and often more or less deformed, as well as on the underground part of the stem and on the enlarged roots themselves, somewhat black, water-soaked lesions of various sizes and appearances may be found. (Pl. 11, A–E.) The decayed spot may occur on one side, or it may cut off the entire food supply by girdling the root. Pits of varying depths with jagged margins one-half inch or more in diameter occur on the swollen roots. The infections apparently have their origin in the small rootlets, the organism growing thence into the sweet potato, the root enlarging about the point of infection, thus leaving a hole, depression, or cavity. (Pl. 11, E.) In the early stages of the disease the lesion is covered by the skin of the root, which later breaks away, exposing a cavity beneath it. In severe cases of infection the enlarged root may be more or less girdled, the root continuing to enlarge on each side of the infected area in the form of a collar, thereby producing a curious disfiguration somewhat crudely resembling a dumb-bell. (Pl. 11, C.)

The number of lesions naturally depends upon the severity of the disease. On the small roots they frequently occur in great numbers, and as many as 25 pits may be counted on a root 5 to 6 centimeters in diameter.

A stunted growth of the vines and roots results from the destruction of the small roots and root hairs. During a dry season the disease is more destructive than during a wet one, a fact which may be due not so much to the more vigorous attack of the organism during dry conditions as to the lessened vitality of the host, caused by an inadequate supply of water. Poole (*137*), in investigations on the relation of soil moisture to infection, found that very little infection occurred in soil containing moisture to the extent of 10 per cent or more of its water-holding capacity, but severe infections occurred in drier soils. His experiments were conducted with two varieties,

[5] United States Department of Agriculture, Bureau of Plant Industry. Crop losses from plant diseases in the United States in 1919. U. S. Dept. Agr., Bur. Plant Indus. Plant Disease Survey Bul. Sup. 12: 325. 1920. [Mimeographed.]

Vineless Yellow Jersey and Triumph. In view of these results, the application of organic material or manure, which would maintain a higher percentage of moisture, should be beneficial.

Causal Organism

Much remains to be learned about the soil-rot organism. Halsted (*61*) in 1890 described the disease as new and named the causal organism *Acrocystis batatae* E. and Hals., a new genus and species. He was unable to obtain infection either in the field or in the laboratory, and no conclusive evidence was furnished to show that he had isolated the organism in pure culture. He describes the disease, however, in some detail and also the morphology of the organism which he believed to be the cause, submitting at the same time several illustrations as proof of what he had seen. Some years later, Taubenhaus (*178*) questioned the conclusions of Halsted, intimating at the same time that the organism studied by Halsted was not the cause of soil rot. He himself had isolated almost exclusively from soil-rot lesions either a Fusarium, a Rhizoctonia, or an Actinomyces.

Two years later Elliott (*47*) published two articles on the disease, the first a preliminary account in which were set forth some of the morphological characteristics of the organism. The second (*46*), much more detailed, not only described the disease and certain inoculation experiments but also the morphology and life history of the causal organism. The results of Elliott were later confirmed by Taubenhaus (*180*), who contributes additional data purporting to show that the soil-rot organism attacks other hosts (potato and beet) as well. He, as well as Elliott, obtained the organism in pure culture. The organism studied by Elliott, which he concluded to be the cause of soil rot, belongs to the Myxomycetes, to which he gave new generic and specific names, without at the same time giving to them technical descriptions, such as good usage at the present times requires. Elliott named this slime mold *Cystospora batata*. Judging from his description of the mode of reproduction and infection, this slime mold presents some very curious and striking differences from other Myxomycetes. He describes (*47*) the organism in part as follows:

> The swarm spores first entering a growing-point go through a rapid development in the outer host cells, passing through an ameboid and a plasmodial stage. During the plasmodial stage a large number of nuclei are formed by mitotic division. The plasmodium then forms a heavy-walled cyst in which hundreds of spores are developed. The swarm spores are liberated within the cyst, which breaks down and releases the spores, when a further infection of host cells occurs. The infection spreads rapidly to the main root, causing a pit or "pox" scar. When the pit has reached the limit of its development the plasmodium assembles and breaks out, migrating into the soil. A secondary infection by swarm spores in small immature pits, causing extensive blisterlike elevations in the skin of stored sweet potatoes, has been observed.

The formation of a heavy-walled cyst containing several hundred swarm spores separates this plasmodium from the now recognized genera of the Plasmodiophorales. Accordingly, the name *Cystospora batata* gen. nov., sp. nov. was proposed for this new organism.

The morphology, life history, and other characteristics of this myxomycete are further detailed in another publication (*46*). Both

Elliott and Taubenhaus emphasize the ease with which this slime mold can be studied, each making a point of the fact that the plasmodia flow out if soil-rot roots are placed in a receptacle in such a way as to provide an abundance of moisture. Curious as it may seem, the writers have been unable to verify the results of Elliott and Taubenhaus, although they have worked on the disease more or less for several years. So far, they have not been able to isolate a slime mold from any of the material studied by them, even when the directions given by Taubenhaus and Elliott are closely followed. They have made hundreds of microscopic slides from sections cut through lesions of all types, showing all stages in the development of the disease, and in no case have they ever observed anything that could be called a plasmodium, cyst, or swarm spore. The sections were stained in different ways designed to bring out such structures. Some of the slides prepared by the writers have been examined by investigators accustomed to the study of Myxomycetes, and the structures reported by Elliott and Taubenhaus in no case have been observed. Several other investigators have also failed to observe a myxomycete on sweet potatoes in moist chambers or to see it in microscopic sections. The writers do not deny the existence of a myxomycete in the soil-rot lesions or its probable cause of the disease; yet they must, however, withhold final acceptance of the results of Elliott and Taubenhaus until more convincing proof has been submitted.

Hundreds of cultures have been made from soil-rot lesions of all kinds which have yielded only a miscellaneous variety of fungi and bacteria. *Fusarium* sp., Actinomyces, and Rhizoctonia have been isolated more frequently than any other fungi. Inoculations with these different organisms have given consistently negative results, which does not mean necessarily that none of them cause the disease, since, as it often happens, failure may be due to the fact that the proper conditions for infection may not have obtained.

Manns (*120*) apparently has not accepted the results of Elliott and Taubenhaus. He has carried on investigations with this disease for several years and has come to the conclusion that it is caused by a species of Actinomyces that resembles *A. poolensis* Taub. Not only has Manns (*120*) isolated this organism from soil-rot lesions, but he has been able to produce "soil sickness" by mixing the contents of several culture dishes into the soil. The results of Manns seem to be fairly conclusive evidence of the connection of Actinomyces with soil rot.

DISSEMINATION

Experiments have demonstrated that soil rot is caused by an organism that lives over in the soil and that can be readily destroyed by sterilization. These results were obtained by experiments with soil from a field that had grown badly diseased sweet potatoes. The soil was divided into three lots, one of which was steamed for four hours at a pressure of about 15 pounds, another was sterilized with formaldehyde in the usual way (1 pint of formaldehyde in 20 gallons of water), and the third lot was held as a control. Sweet potatoes were planted in the three lots in an outdoor bed and allowed to grow during the summer. In the fall the potatoes were carefully examined

when harvested. Those from both lots of sterilized soil were entirely free from soil-rot lesions, while those from the untreated soil were badly pitted.

Apparently the disease is not generally transmitted through the potatoes used for seed. Evidence to this effect was obtained in experiments several times repeated, in which a number of badly pitted soil-rot roots were bedded in noninfested soil. After the sprouts were well developed they were planted in a field where soil rot has never occurred. The sweet potatoes in the fall were entirely free from soil rot.

There is a possibility that the sprouts when grown in infested seed beds may carry the disease to the field, although experiments have failed to demonstrate this fact. It seems possible, however, that the causal organism may be carried to the field in the soil clinging to the roots of plants grown in infested soil.

Dissemination, so far as the writers are able to determine at the present time, is brought about principally by the transportation of the soil, which may be done in several ways—i. e., by the blowing of the soil during high winds, by means of drainage water, by farm implements, or by livestock that roam from one field to another. The dissemination over long distances is probably brought about by the shipment of sweet potatoes with infested soil adhering to them.

MOTTLE NECROSIS

History

Mottle necrosis (caused by *Pythium ultimum* Trow and *P. sclerotiechum* Drechsler) was recognized as a distinct disease of sweet potatoes in 1917, although it is barely possible that it was seen by Halsted (*61*) in 1890 and described under the name of "white rot." Halsted's investigations were somewhat meager, but his descriptions of the characteristics of the disease itself and an organism associated with it indicate that he was describing the disease known to-day as mottle necrosis. Information has been obtained from growers in New Jersey that mottle necrosis has been known to them for many years. It appears that since it occurred only intermittently and the losses were relatively small that no serious attention was given to it. Since 1917 there have been frequent reports of its occurrence in various localities where susceptible varieties are grown.

Geographical Distribution and Economic Importance

Mottle necrosis was collected for the first time on the Yellow Jersey variety at the Arlington Experiment Farm, near Rosslyn, Va., in 1917. The next year it was collected by Lauritzen in North Carolina. Since then specimens have been collected or received from New Jersey, Delaware, Virginia, Maryland, South Carolina, and Mississippi. Inasmuch as the disease is already known in widely separated regions, it is not unlikely that it occurs in other States. The Yellow Jersey, Big-Stem Jersey, Triumph, and to a limited extent the Gold Skin varieties, have been found infected under natural conditions. The Triumph is the only one of these varieties grown to any large extent in the South, and so far mottle necrosis has not been reported on this variety except when grown in the North for experimental purposes.

It is possible that climatic conditions may be such as to restrict its extensive development in the South.

No extensive losses have been attributed to mottle necrosis except in a few fields in New Jersey and in one locality in Virginia. In New Jersey as much as 10 to 40 per cent of the crop has been destroyed on some farms. The disease occurs each year on certain light sandy soils of the Arlington Experiment Farm, near Rosslyn, Va., and losses as high as 50 per cent are not uncommon.

Symptoms

Mottle necrosis is a field disease and is not likely to be confused with any of the others, with the possible exception of soft rot. It can, however, readily be distinguished from soft rot by isolation of the fungus from necrotic tissue and usually by a microscopic examination of macerated cells.

Superficially, mottle necrosis may be quite inconspicuous. The surface lesions (pl. 12, A) vary from small, dirty-brown, somewhat sunken spots (*82*) at the base of small rootlets to conspicuous sunken areas of various sizes and shapes. The presence of relatively inconspicuous lesions on the surface does not necessarily indicate that the decay within is small in extent.

There are three types of mottle necrosis, which for the sake of convenience have been designated the band, marble, and cheesy types. The first of these is by far the least common and is characterized by a band of dead tissue 1 to 3 mm. thick, outside of and rarely penetrating the fibrovascular ring, extending around the potato. The marble type (pl. 12, C) is best seen in a section through the root and is more prevalent in late summer and early fall. It is characterized by the irregularly shaped islands of necrotic tissue of various sizes scattered more or less indiscriminately through the roots. The organism ramifies from the point of infection in various directions, forming chambers, or pockets, which might be assumed from a casual examination to be disconnected and to have no common place of origin. The tissue is dry, crumbly, and varies in color from a dark gray to chocolate brown.

In a typical example of the cheesy type (pl. 12, B), as found in the field or as produced by artificial inoculation, the tissue is of about the consistency of soft cheese and of a grayish color. Instead of the fungus ramifying in many directions and forming almost isolated pockets, large chambers with comparatively smooth walls are produced.

All three types of decay are produced by the same organism. If decay takes place at a relatively high temperature the marble type will result. If the temperature is comparatively low the cheesy type will occur. The latter occurs in the late fall when the field temperatures are rather low.

Causal Organisms

Inoculation experiments (*77*, *95*) have demonstrated that three species of Pythium, *P. scleroteichum* Drechsler, *P. ultimum* Trow, and *P. debaryanum* Hesse, isolated from sweet potatoes are pathogenic. Another species, *P. aphanidermatum* (Edson) Fitzpatrick, the cause of nesting in beans in transit (*94*), was proved to be a rapid

grower, a virulent parasite of sweet potatoes, and to produce typical mottle necrosis. Species from other hosts have been tried on sweet potatoes, but the results were somewhat doubtful. Attention should be called to the fact that *P. ultimum*, the species mostly responsible for rootlet rot of sweet potatoes, is the one commonly isolated from mottle-necrosis potatoes in the field.

PATHOGENICITY

No detailed account need be given of the hundreds of inoculation experiments conducted to prove that several species of Pythium may cause mottle necrosis. Two of these, *P. ultimum* and *P. sclerotei-chum*, stand out conspicuously with respect to their prevalence. All varieties of sweet potatoes have been infected artificially, although only a few (Yellow Jersey, Triumph, Big-Stem Jersey, Gold Skin, and occasionally Belmont) are infected naturally in the field. The results, however, show that all varieties are potential hosts to these parasites.

A study of the relation of temperature to infection and extent of decay caused by *Pythium ultimum* in incubators with controlled temperature showed that the optimum temperature for the growth of the mycelium in pure cultures on corn-meal agar is approximately 32° C., but the greatest prevalence of decay occurred at a temperature of 12° to 15° C.

DISSEMINATION

So far as known, there is nothing distinctive about the dissemination of Pythium. Species of Pythium occur in almost every soil and on a great variety of hosts, and to find them in any soil or on any plant parts would occasion no surprise. Pythium rootlet rot occurs every year to a greater or less extent in the hotbed, so that even though it did not occur in the field it would be introduced there with the plants. It must not be assumed, however, that the planting of sprouts infected with rootlet rot will be followed by mottle necrosis in the field. Diseased sweet potatoes would probably not be employed for seed, and if they were they would not transmit the disease, since it has been shown that Pythium survives only a short time in decayed sweet-potato tissue.

ROOTLET ROT

Rootlet rot is primarily a disease of the rootlets (pl. 12, D) in the seed bed, although the causal organism (*Pythium ultimum* Trow) can be isolated frequently from the small roots of plants in the field throughout the entire growing season. Rootlet rot causes the death of 1 to 3 centimeters of the root ends and is often associated with Rhizoctonia both in the hotbed and in the field.

Rootlet rot occurs in almost any type of soil, but is worst in fairly wet beds containing a considerable proportion of organic matter. It may occur, however, in beds made of almost pure sand.

Its causal organism (*Pythium ultimum*) is one of the species causing mottle necrosis. Occasionally other species of Pythium are isolated from the decayed root ends.

A careful examination of 21 varieties of sweet potatoes in the hotbed showed that the disease occurs on all of them (*76*), but not to

the same degree. The varieties can be divided roughly into three groups, according to the degree of infection, as follows: Severely infected—Big-Stem Jersey, Key West Yam, Creola, Yellow Jersey, Nancy Hall, and Porto Rico; moderately infected—Red Jersey, Belmont, Triumph, Yellow Yam, Gold Skin, Dooley, Haiti, Dahomey, Red Brazil, General Grant Vineless, Pierson, and Southern Queen; slightly infected—Pumpkin Yam, Yellow Strasburg, and White Yam. This classification is based on an estimate made when the plants were set in the field. Several varieties classed as moderately infected were on the border line of the slightly or severely infected groups.

It is interesting to note that one of the varieties (Triumph) that is very susceptible to mottle necrosis is classed as only moderately susceptible to rootlet rot. Under field conditions, on the other hand, such varieties as Pumpkin Yam, Yellow Strasburg, and White Yam, while very susceptible to rootlet rot, are not susceptible to mottle necrosis.

SCLEROTIAL BLIGHT

Geographical Distribution and Economic Importance

Sclerotium rolfsii Sacc. causes diseases of a large variety of crops besides sweet potatoes in the United States (*131*, *181*, *145*, *63*), as well as in the tropical and semitropical world (*24*, *205*, *161*). Many of these crops are grown in rotation with sweet potatoes, and most of the soil in the southern part of the sweet-potato belt is infested.

The losses to sweet potatoes caused by *Sclerotium rolfsii* are restricted mostly to the seed bed, although a few cases of injury in the field have been noted by Peltier in southern Alabama. It is not surprising that the organism should attack the plants in the field, in view of the fact that the fungus is parasitic on many other crop plants (*43*, *44*, *116*, *169*) under similar conditions. Observations made on this disease in seed beds, as well as experiments with the fungus in the greenhouse, indicate that the organism causes most damage at high temperatures and a relatively high humidity.

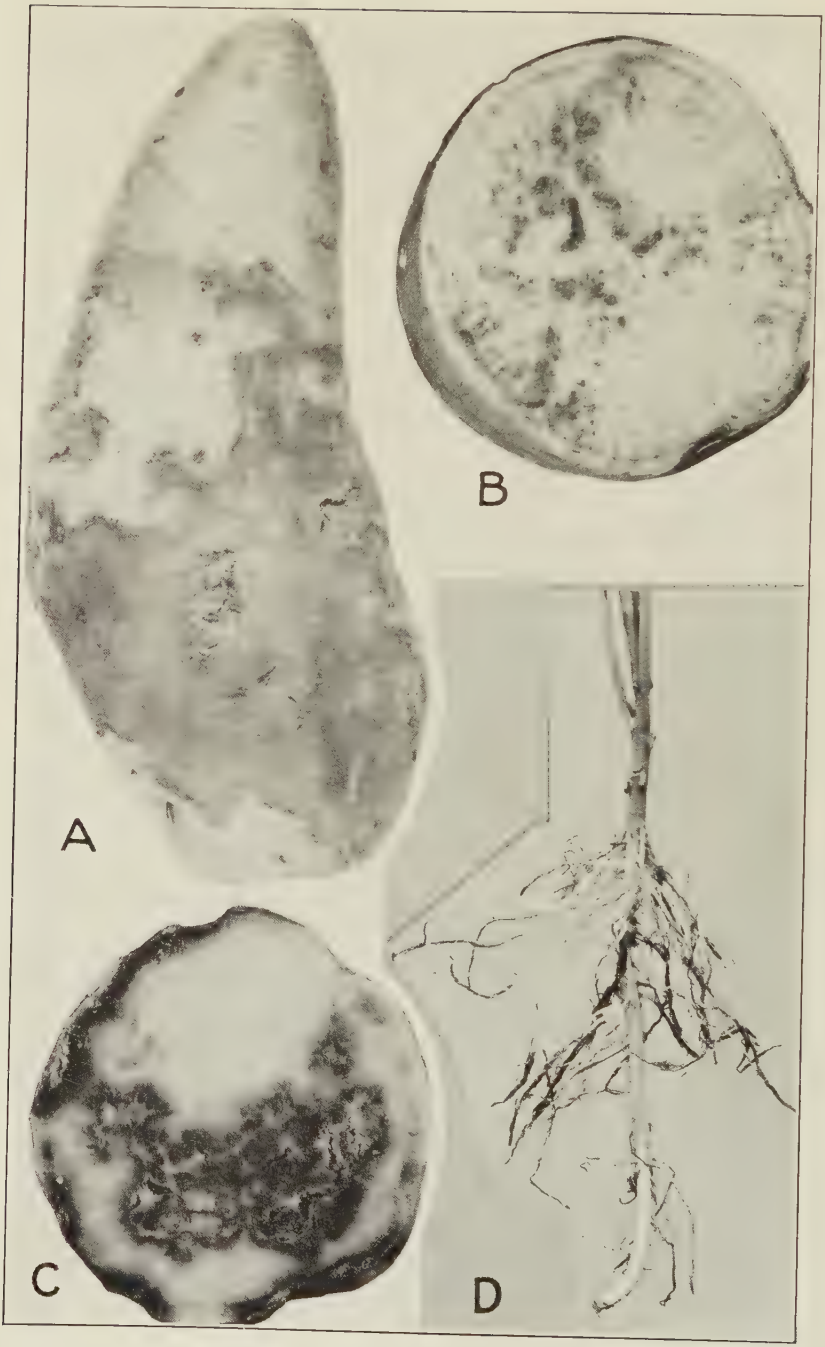
Sclerotial blight is quite common in the open seed beds in Florida, Texas (*74*), Alabama, and Mississippi (*126*), and in most if not all of the Gulf Coast States, especially following periods of rainy weather. There are, nevertheless, rainy periods in the spring with high temperature when the fungus spreads from centers in the beds and kills the plants in spots of considerable size.

In many places in the Southern States the plants are forced by the application of heat beneath the plant bed, the heat and moisture being increased by a covering of canvas or by other means. *Sclerotium rolfsii* flourishes under such conditions, and in some seasons it may cause considerable loss. In the covered beds there may be many infection centers from which the fungus spreads. The removal of the canvas or covering at least a portion of the day, thereby lowering the humidity, will reduce the loss somewhat.

O. C. Boyd advised the writers by correspondence that in Georgia *Sclerotium rolfsii* may cause a storage rot of sweet potatoes in banks when the water seeps in and wets the potatoes. The writers have infected the fleshy roots by artificial inoculation.

SOIL ROT

Progressive stages in the development of the disease are shown from A to E. Note at C the constriction near the center of the root



DISEASES CAUSED BY PYTHIUM SPP.

A.—External symptoms of mottle necrosis (caused by *Pythium ultimum* and *P. scleroteichum*). B.—Cheesy type of decay of mottle necrosis. C.—Marble type of mottle necrosis with indications of the band type. D.—Rootlet rot (caused by *P. ultimum*)

SCLEROTIAL BLIGHT (SCLEROTIUM ROLFSII)

A.—These plants were inoculated by placing mycelium in the soil about them. They were then held in an inoculation chamber with a high humidity and high temperature. B.—Half of a wounded sweet potato; the cut surface is partially decayed with *Sclerotium rolfsii*. Sclerotia are produced in abundance

RHIZOCTONIA ROT (CORTICIUM VAGUM)

A and B.—Conspicuous cankers on the roots and underground portions of the stem. C.—Basidial stage on the upper portion of the stem

Although sclerotial blight may occur on sweet potatoes in the field, it is generally of no consequence under such conditions, the greatest loss being restricted to the seed bed. It has never been observed in storage houses, and it probably does not cause much damage there. The potatoes are difficult to infect artificially, although in the hotbeds the mycelium has been found repeatedly on the mother potatoes.

Symptoms

Sclerotial blight on sweet potatoes in the seed bed usually begins as small infection centers during wet warm weather. The stems of the plants are rotted away just at, or below, or above the soil, the fungus producing a soft brownish rot resembling in general appearance damping off, caused by this and other fungi. The earliest symptoms are characterized by a yellowing of some of the lower leaves and a stunting of the plant, which, if pulled gently, will separate easily from the mother potato. At the bases of the stems and on the ground about the plants a moldy growth of white mycelium is produced, in which is intermixed a large number of sclerotial bodies in all stages of maturity. If the plants are rather large and thick, thereby maintaining an abundance of moisture about the stems and on the soil, the mycelium grows upon the plants and over the surface of the soil, spreading radially from plant to plant and eventually injuring or destroying plants over areas of considerable size, sometimes several feet in diameter.

Causal Organism

Sclerotial blight is caused by the sterile fungus *Sclerotium rolfsii* described by Saccardo (*150*). So far as known, there is no fruiting stage in its life history, reproduction being accompanied wholly by means of the hyphae and the sclerotia. The hyphae are somewhat coarse, the cells being 2 to 9 μ by 150 to 250 μ, and, according to Higgins (*98*), characterized by the presence of clamp connections and a peculiar method of branching, which he claims are valuable aids in identifying the fungus. The sclerotia, at first white, becoming brown with age, are more or less smooth and glossy and somewhat resemble mustard seeds. They are one-half to 1½ millimeters in diameter and are composed of interwoven hyphae. As long as a fruiting stage is unknown, the exact taxonomic position of the fungus can not be determined. Higgins, as a result of morphological studies, claims that the affinity of *S. rolfsii* to the class Basidiomycetes is indicated by the septate, binucleate mycelium and by the presence of the clamp connections at the septa.

The infection experiments carried out by Fulton (*55*), Wolf (*208*), Taubenhaus (*182*), Harter (*73*), and others clearly demonstrate that *Sclerotium rolfsii* is a parasite on a number of different hosts, among them the sweet potato. The writers found that young sprouts of various sizes and of different ages were destroyed in the seed beds if hyphae and sclerotia were mixed in the soil, provided the moisture and temperature were kept sufficiently high. Young unwounded plants just coming through the soil were more readily attacked than older ones, and if they were confined in an infection cage all the plants were practically destroyed in two to five days. If, on the other

hand, they were left exposed to outdoor conditions or to the environment of an ordinary greenhouse infection usually did not result.

LIFE HISTORY

Seed potatoes are often covered with the mycelium of *Sclerotium rolfsii* in the seed bed, and it is doubtless by this means that many plants become infected. (Pl. 13, B.) The fungus spreads also through and over the soil and thus comes in contact with and infects other plants.

That both high temperature and high relative humidity may be necessary for the best development of *Sclerotium rolfsii* was suggested by experiments in the greenhouse as follows: Sound potatoes were bedded in small boxes filled with soil sterilized by steam for one hour at a pressure of 20 pounds. Before the plants came up and after they were 3 to 5 inches high pure cultures of *S. rolfsii* were mixed in the soil. Although the humidity under greenhouse conditions can not be said to be especially high, the temperature reached from 85° to 90° F. in the daytime. No infection occurred in three weeks. Later these same boxes were removed to an infection cage in the same greenhouse, where the humidity was considerably increased by thoroughly wetting the inside of the boxes containing the plants. The cage was then tightly closed. In two days a white mycelial growth of *S. rolfsii* partially covered the soil and plants, and in two more days some of the plants were nearly rotted off at and a little above the soil line; some had begun to topple over. (Pl. 13, A.) By this time the coarse strands of the hyphae intermixed with sclerotial bodies in all stages of maturity had spread farther over the soil and plants.

In localities where *Sclerotium rolfsii* had been observed in the seed beds the fungus has been found in abundance in the plants prepared for shipment. In southern Alabama the Porto Rico variety seems to be especially susceptible, and the Triumph much less so.

In the absence of spore forms, which in the case of *Sclerotium rolfsii* appear to be lacking, the perpetuation of the fungus must be brought about by means of the mycelium and the sclerotia. The sclerotial blight organism occurs quite generally throughout the tropical world and as far north as the District of Columbia and Illinois in the United States, most of which region is comparatively warm throughout the year. Undoubtedly, in some of this territory the fungus and its host plants grow throughout the year. *S. rolfsii* also produces an abundance of sclerotia, which are better adapted to withstand unfavorable weather conditions than the mycelium. The fact that the fungus does not occur in the Northern States to any extent may be due, as Higgins suggests (*98*), to the inability of the sclerotia to last long when brought in contact with moist soil under field conditions and also, as he further points out, to its very great susceptibility to freezing when wet. On the other hand, if kept dry in a culture tube it will grow readily when transferred to fresh media when more than 2 years old.

RHIZOCTONIA ROT

Rhizoctonia rot caused by *Corticium vagum* B. and C. (*Rhizoctonia solani* Kühn) occurs commonly in the seed bed and has been

found occasionally in the field. It is more prevalent in some seasons than in others, depending to a large extent on weather conditions.

Rhizoctonia, only recently recognized as the cause of a disease of sweet potatoes (*74*), like *Sclerotium rolfsii*, occurs on a large number of hosts.

Geographical Distribution and Economic Importance

Rhizoctonia rot was observed on sweet-potato plants in the hotbeds in a number of the Southern States and in New Jersey in 1915. Since then it has been observed repeatedly on plants in seed beds throughout the entire sweet-potato belt. Poole reported the occurrence of *Rhizoctonia* sp. on the Nancy Hall, the only one of 11 varieties bedded in the greenhouse affected. The sclerotia formed on the surface resembled those produced on the potato. The sprouts were not attacked. Burger (*18*) reports that Rhizoctonia caused a damping off of sweet potatoes in Florida in the seed bed.

On the whole, Rhizoctonia rot is of no great economic importance, although 5 per cent or more of the plants have been destroyed in the seed bed in a few isolated cases. It has been found occasionally in the field on the roots of plants growing in sandy soils in Washington, Delaware, New Jersey, and Virginia, and it is probable that it occurs elsewhere. Rhizoctonia is frequently found associated with Pythium on the small roots and on the rootlets. The small roots are more or less completely decayed from the tips to the main root. Following the destruction of the small roots the plant may develop a large number of new roots, producing a condition somewhat resembling a witches'-broom (*95*), or as frequently happens in impoverished soil no new roots are formed, there being nothing left except the central root with a few rootlets partially or entirely dead. In either case the plants remain stunted, producing no potatoes or only a very few small ones.

Rhizoctonia may cause one or more cankers of various sizes on the stem, which do not seem to develop further or cause any injury when the plants are set in the field, as is shown by the following experiment: Several hundred plants with one or more such cankers were planted in the field and kept under observation during the growing season. After six weeks the cankers had entirely disappeared. Observations were made again when the crop was harvested, and no trace of Rhizoctonia injury was found.

Symptoms

It is possible to identify three types of injury caused by Rhizoctonia, one of which is characterized by the production of cankers 2 to 6 millimeters in diameter (pl. 14, A and B) on the stem. These cankers may occur at any place on the plant from the point of attachment to the potato to an inch or more above the surface of the soil. Another type of injury in which there are numerous dead spots or cankers on the rootlets sometimes occurs. What seems to be a third type of injury, found more prevalent and destructive in Alabama and other Southern States, is characterized by a complete rotting away of the rootlets and even the underground part of the stem. There has always been some question as to whether the destruction as represented by the third type is due to Rhizoctonia

alone. In the rotted underground portion of the stem other fungi were found, but Rhizoctonia was also present in considerable abundance.

Causal Organism

The Rhizoctonia rot of sweet potatoes is caused by the fungus *Corticium vagum* B. and C. (*Rhizoctonia solani* Kühn). The basidial stage has been found in a few instances in commercial beds. A white powdery growth (pl. 14, C) characteristic of this stage was conspicuous on the stems and lower leaves near the soil in a bed in the greenhouse in 1917, where there was a heavy growth of foliage and a relatively high humidity. Cultures made from the basidiospores produced a characteristically constricted and septate mycelium identical with that found in cultures from cankers on the stems. (Fig. 10.) The spore cultures were obtained as follows: Infected plants were brought to the laboratory and suspended in the evening over a plate of potato agar. The following day a small square of the potato agar on which spores had germinated was transferred to a plate of Beijerinck's agar, on which a few sparsely branched hyphae were produced. One of these hyphae was then marked out in the plate and a section of it cut out and transferred to another plate. By this method a pure strain, which was regarded as equivalent to the isolation of a single basidiospore, was obtained.

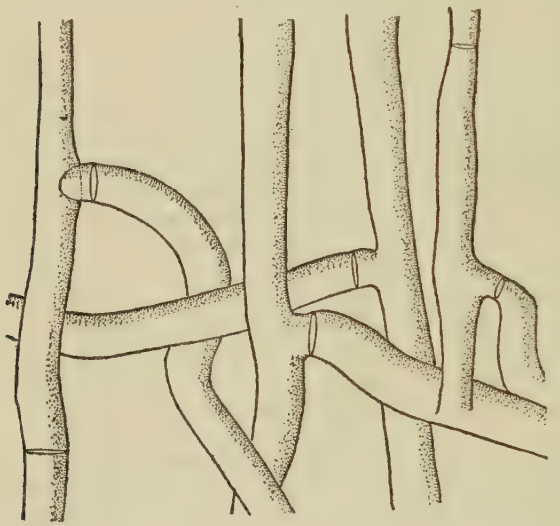


FIG. 10.—Mycelium of the Rhizoctonia stage of *Corticium vagum*. × 1,600

The shape of the basidiospores, the character of the hyphae, septations, and other morphological characteristics of the fungus (fig. 11) are no different from those found by other investigators (*130*, *40*) for the same fungus from other hosts. The spores are borne singly on sterigmata of which there are four on each basidium. The spores germinate by the development of one or two germ tubes, a septum being laid down in the germ tube a short distance from the spore. Large sclerotialike bodies were produced in culture, but none have ever been found on the host.

There seems to be a difference in the size of the basidiospores between the strain from sweet potato and the strain studies from the potato by Rolfs (*144*), who found them to vary from 6 to 13 μ by 9 to 15 μ. A number of measurements made by the writers in which no extremes were purposely selected showed the basidiospores from sweet potato to be somewhat smaller (3.5 to 5.5 μ by 7.5 to

9.5 μ). It is probable that if extreme sizes had been looked for considerably larger and smaller spores would have been found. It is possible that a variation in the size of the basidiospores may be characteristic of the fungus on different hosts.

WHITE RUST

History

The white rust due to the fungus *Albugo ipomoeae-panduranae* (Schw.) Sw. was first described on the sweet potato by Halsted (*61*) under the title "Leaf mold." Since then there have been frequent reports of its occurrence and distribution in the United States and in foreign countries. It is said to be a common parasite on many species of Ipomoea.

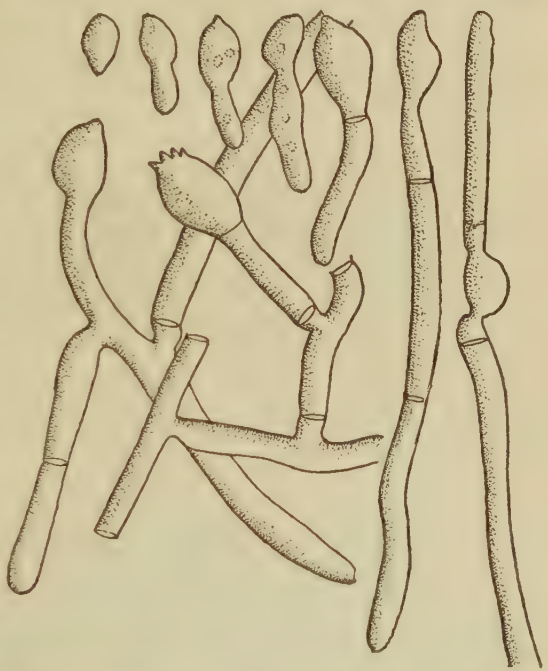


FIG. 11.—Basidial stage of *Coniothecium vagum*, showing basidia and germinating basidiospores. × 1,100

Geographical Distribution and Economic Importance

The white rust is very widely distributed in the United States and foreign countries, having been collected by the writers in nearly all parts of the United States where sweet potatoes are grown. It is quite prevalent in some seasons, especially following a period of several days of rainy weather accompanied by cool nights. White rust occurs abundantly in some of the Southern States and along the Gulf coast, but because of the prevailing dry weather and warm nights it has not been very prevalent in Texas, Oklahoma, and some of the other Western States. It is prevalent each year in Cuba and in some of the other islands of the West Indies. Ashby (*8*) reports a white rust (*Albugo* sp.) on sweet potatoes in Jamaica, but he apparently did not determine the species, and Stevenson (*171*) lists it among other fungi on sweet potatoes in Porto Rico. Nowell (*128*) also found the same disease on sweet potatoes in the West Indies. White rust has been reported in Haiti (*60*), in Argentina by Hauman-Merck (*97*), in Brazil by Sydow (*175*), in Trinidad by Rorer (*146*), and in Guam by Weston (*206*). Weston reported only slight loss in Guam. From the published reports it would seem

that this disease is much worse in the United States and the islands of the West Indies than in any other part of the world. This may not necessarily be the case, however, since its occurrence may have been more uniformly noted and reported from America than elsewhere. In practically no case is the white rust destructive enough to occasion any considerable loss.

Symptoms

Infection by white rust usually takes place on the under side of the leaf, probably through the stomata, although there may be few or many lesions (pl. 15, A and B) on any of the aboveground parts of the plant. Early infections are characterized by a yellowing caused by a partial loss of chlorophyll about the infection court and on the upper side of the leaf directly over it, or by the malformation (pl. 15, B) and distortion of the leaves, petioles, and succulent stems or by both. The disease spreads and develops rapidly when the

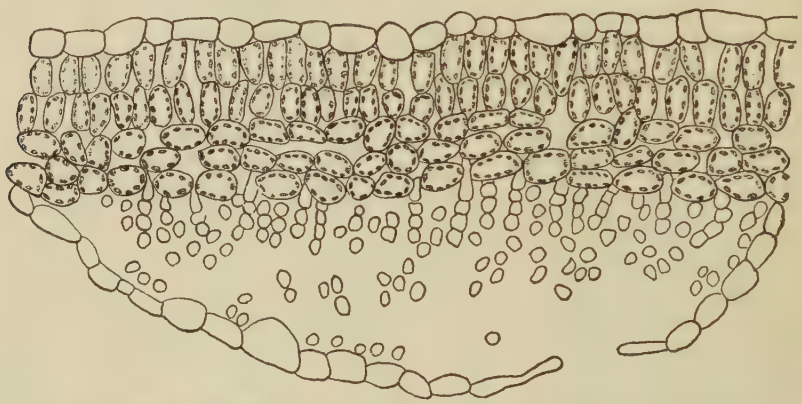


Fig. 12.—Semidiagrammatic drawing of cross section of sweet-potato leaf, showing a white-rust pustule. × 135

humidity is high and the nights cool. If such weather is followed by warm dry days the infected leaves may be killed, dry up, and fall off. Pustules (fig. 12) are produced beneath the surface of the epidermis, which is finally ruptured by pressure from below, and the spores escape.

Causal Organism

The organism causing white rust of the sweet potato was first called *Cystopus ipomoeae-panduranae* by Halsted, but, in accordance with more recent usage, it later became known as *Albugo ipomoeae-panduranae* (Schw.) Sw.

The conidia, which measure 12 to 22 by 10 to 21 μ, are borne in chains in great numbers in the pustules. (Fig. 12.)

Artificial inoculation of sweet-potato leaves gave negative results in most cases. The experiments, however, resulted in developing the fact that special conditions are probably necessary to bring about infection. One set of experiments in which the conidia from freshly gathered leaves were smeared on the under side of the leaves of

plants growing in the greenhouse resulted in a few infections. Other similar experiments in the greenhouse and in infection cages where a high humidity was maintained gave negative results, even though the conidia were germinated before they were sprayed on the leaves. Plants were also sprayed with germinated conidia and placed at temperatures of 12° to 14°, 14°, 20°, and 25° C. and held there for 36 hours, but in no case was there any infection.

On August 9, 1917, germinated conidia were sprayed on several plants grown near Rosslyn, Va. The plants were covered for 24 hours with large glass bell jars, which in turn were wrapped in coarse manila paper. In about 10 to 14 days there was an outbreak of white rust in the immediate vicinity of the sprayed plants, other patches approximately 100 yards away remaining entirely free from the disease during the summer.

From the result of other experiments there are reasons to believe that certain favorable conditions, especially cool nights, are necessary for the germination of the conidia and consequent infection. It has been shown by Melhus (*123*) and others for other members of the

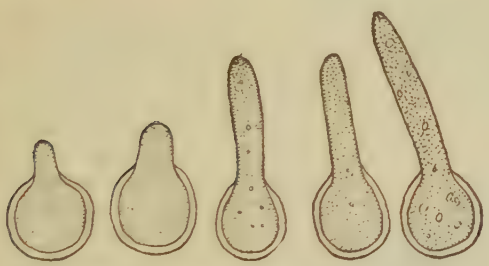


FIG. 13.—Direct germination of the conidia of *Albugo ipomoeae-panduranae*. × 1,200

FIG. 14.—Formation of zoospores in conidia of *Albugo ipomoeae-panduranae* and their subsequent germination. × 1,200

Peronosporaceae that chilling temperatures stimulate germination of the conidia. The writers made no attempt to establish the minimum, optimum, and maximum temperatures for germination, but found that only rarely did the spores germinate at room temperature. The experiments had for their object to determine whether chilling temperatures were necessary for the germination of the conidia. It was found that when the conidia were scraped from the leaf into tap water and then placed at a temperature of 12° to 14° C. they would germinate abundantly in 30 to 45 minutes. This temperature was therefore used to test spore germination in all the other experiments which followed. At all temperatures ranging from 12° to 25° germination was complete in one and one-half hours. No germination occurred in the same length of time at a temperature above 25° or below 8.2°, although it is not unlikely that if the spores had been held at the lowest temperatures for a longer period of time some germination might have taken place.

The conidia sometimes germinate by the direct production of a single germ tube (fig. 13), but more frequently 4, 6, 8, 10, and as high as 16 zoospores were produced. (Fig. 14.) All the zoospores usually escaped from the conidia at about the same time, though occasionally

one or two would emerge in advance of the others. After escaping from the conidia the zoospores remained quiescent for a moment and then set up a violent rolling or jerky movement. Soon thereafter they pulled apart and swam hastily around in the water for a short time and then rounded up and became nonmotile. The time consumed by the zoospores in emerging from the conidia and in separating from each other varied from one to three minutes. The zoospores germinated (fig. 14) in about two and one-half hours after the conidia were placed at a temperature of 12° to 14° C.

Two whiplike flagella, from one and one-half to two and one-half times as long as the diameter of the swarm spore, are attached to it at nearly the same place. The flagella can be shown beautifully by staining with a dilute solution of eosin in alcohol by the following method: Very young, active, motile zoospores should be mounted on a microscopic slide in a small quantity of water. As soon as the swarm spores separate and while they are still actively motile, pass a little eosin in alcohol under the cover slip. In a few moments the swarm spores come to rest, and the outline of the whiplike flagella can easily be observed.

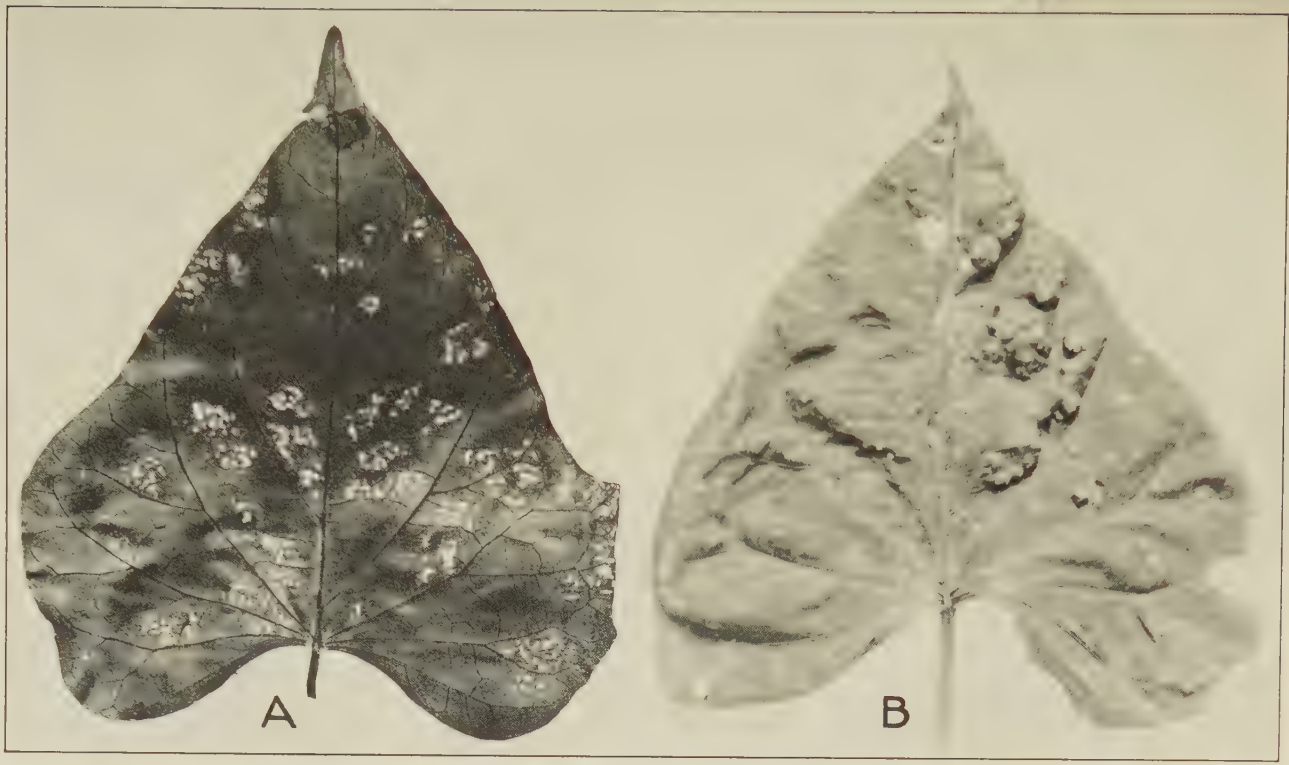
LEAF BLIGHT

The occurrence of leaf blight, caused by *Phyllosticta batatas* (Thüm.) Cke., is more or less correlated with weather conditions, and if the season is favorable for its development it may be present in abundance. In some parts of the United States it occurs to some extent each year, while in others it may be entirely absent, particularly in the more arid portions of the sweet-potato growing region.

Its distribution in the United States is almost as extensive as that of the sweet-potato crop, the disease being more prevalent along the South Atlantic seaboard, the region along the Gulf of Mexico, and in Cuba. At the present time very little is known about the distribution of this disease outside of the United States. The writers have collected it in Cuba, and Stevenson (*171*) reports its occurrence in Porto Rico.

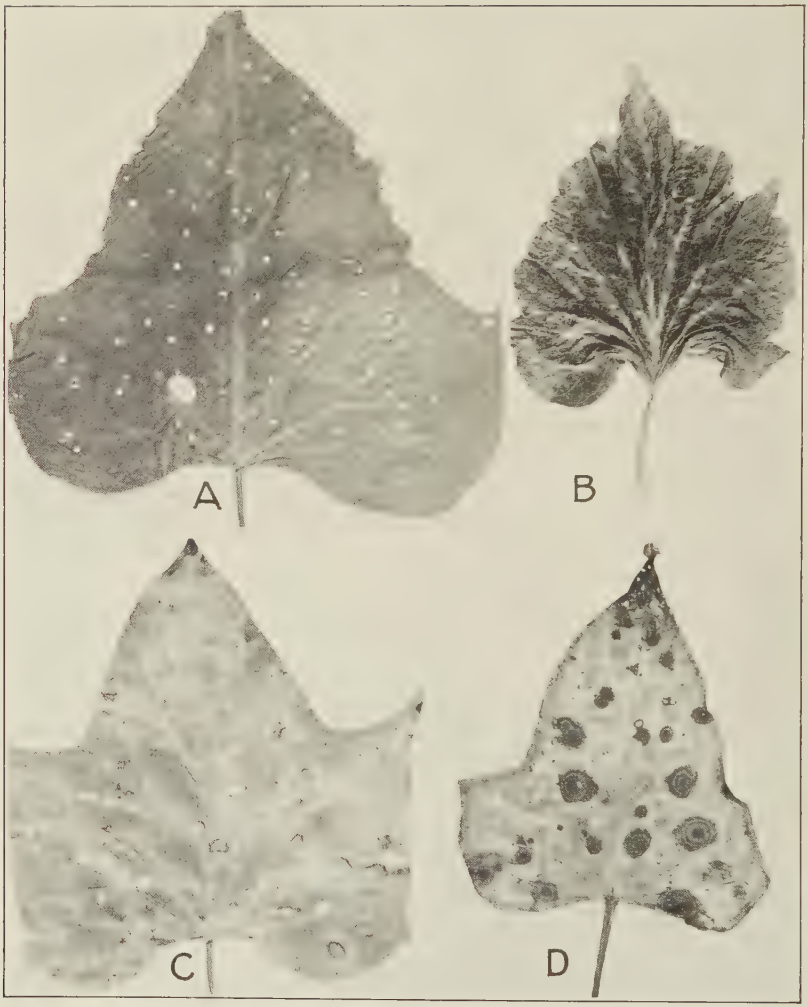
Symptoms

Leaf blight causes spots of irregular shape to nearly circular from 3 to 8 millimeters in diameter on the upper surfaces of the leaves. (Pl. 16, C.) The center of the spots, which are studded with the pycnidia, may be either light gray or cinnamon brown. The difference in color of the lesions is not regarded as significant, one color probably being merely a stage in the development of the other. Both types of spots have been found on the same leaf, and so far as the writers have been able to determine, there is no difference in the size and shape of the pycnidia and spots produced by them. The spots with cinnamon-brown centers appear to be younger than those with gray centers and develop into the latter. The spot is circumscribed by a slightly raised border. (Pl. 16, C.) Sometimes, though not always, the tissue for 3 to 10 millimeters outside the border develops a decidedly conspicuous purplish color, the production of which has been more pronounced in the southern part of the United States and in Cuba than elsewhere. No attempt has been made to seek an explanation as to why a purple color is developed around these



WHITE RUST (ALBUGO IPOMOEAE-PANDURANAE)

A.—View of the lower surface of a sweet-potato leaf, showing the numerous infections of white rust. B.—Malformations of the leaf which frequently accompany infections by this fungus

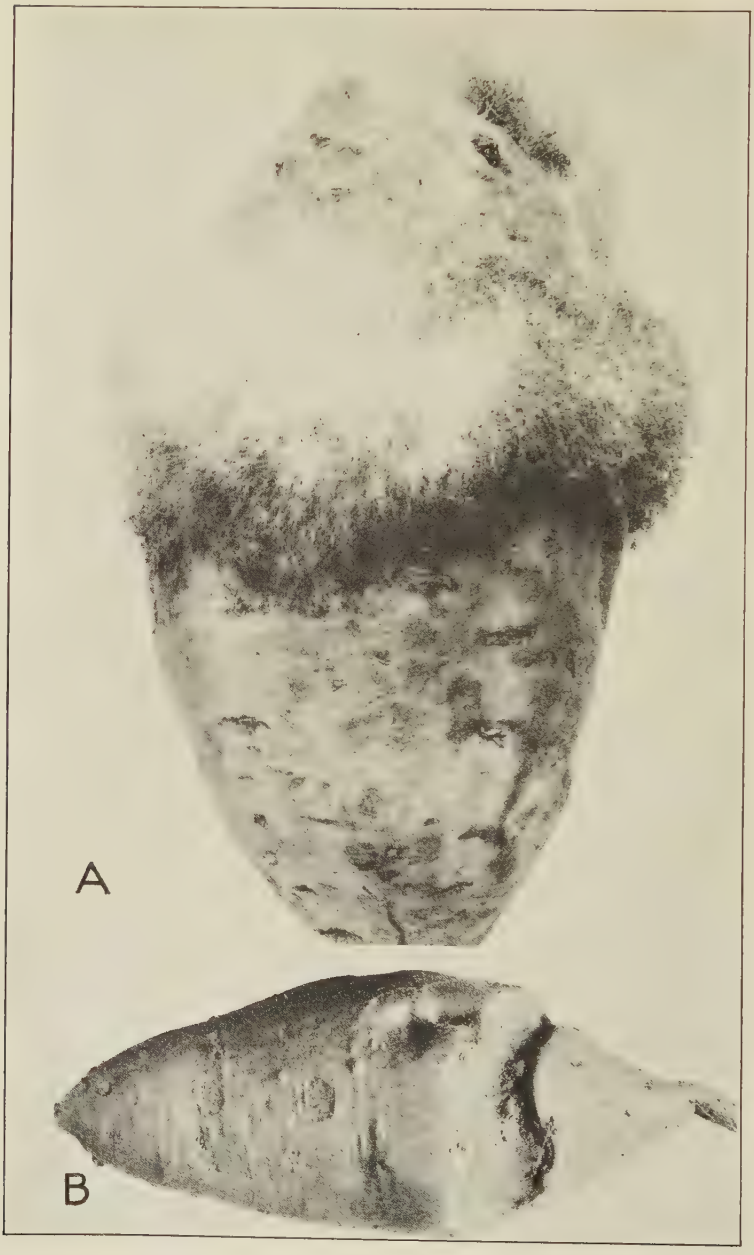


MISCELLANEOUS LEAF DISEASES

A.—Sweet-potato leaf showing a natural infection (white spots) by *Septoria bataticola*. Note that the center of some spots is broken out. B.—Leaf of sweet potato, showing mosaic. C.—Leaf blight caused by *Phyllosticta batatas*. Note the numerous pycnidia in the dead tissue of the spots. D.—Alternaria leaf spot. The Alternaria develops on the upper surface of a leaf over tissue injured by the white rust on the under side

MISCELLANEOUS STEM AND LEAF TROUBLES

A.—Three leaves that show progressive stages of sun scald. B.—Two plants upon which *Fuligo violacea* is growing. C.—Typical example of flat fasciation



TWO ROTS CAUSED BY THE BREAD MOLD

A.—Soft rot (*Rhizopus nigricans*). If the skin is ruptured the mycelium develops on the surface, producing numerous sporangia. B. - Ring rot (*R. nigricans*). The point of infection is somewhere between the two ends of the potato, and the progress of the decay is in the form of a collar

spots or why it is more pronounced at some places than at others. Some of the sweet-potato varieties grown in the South apparently contain a considerable quantity of anthocyanin, if one may judge from their purple stems and petioles. If such is the case, it is possible that the purple band may be the result of the destruction by the fungus of the chlorophyll, which normally masks the anthocyanin.

Causal Organism

The causal organism was first described by Thümen[6] as *Depazea batatas* from specimens collected in South Carolina and later by Cooke and Ellis (*32*) as *Phyllosticta batatas*, also from material collected in the United States. Cooke indicates that the fungus described by him is the same as the one described by Thümen. Ellis and Martin (*53*) in 1882 described the fungus as *P. bataticola*, which Ellis and Everhart (*52*) regarded later as synonymous with *P. batatas*.

The pycnidia are more or less dome shaped and vary in diameter from 100 to 125 μ.

The hyaline one-celled spores are ovate, oblong, or kidney-shaped (fig. 15) and may or may not contain one or two oil droplets.

Phyllostictas, evidently saprophytes or secondary invaders, are commonly found on leaves and dead and moribund vines, as well as on stems, injured by the sun, white rust, or other agencies. In some cases the spores are both shorter and narrower than those of *P. batatas*, while in others they are about equal in length and much narrower. The following measurements of what appear to be saprophytic forms show the variation in the spores from the different sources: Cuba, dead sunburnt areas on the leaves 2.6 to 4.1 by 1.7 to 2.6 μ; Florida, 5.8 to 10.0 by 1.7 to 3.1 μ; the upper surface of the leaves from New Jersey over an area injured by white rust, 5.2 to 6.9 by 2.6 to 3.4 μ. On the other hand, the spores of *P. batatas* collected from different sources measured as follows: Virginia, 5.1 to 8.5 by 2.5 to 4.2 μ; Georgia, 3.4 to 9.3 by 2.6 to 5.8 μ; Cuba, 5.2 to 8.6 by 2.8 to 4.1 μ.

Fig. 15.—Conidia of *Phyllosticta batatas*. × 1,200

Phyllosticta batatas does not cause enough damage to require remedial measures. So far as known, it has no other host. Nearly all, if not all, varieties of sweet potatoes are susceptible to the disease.

MINOR FIELD DISEASES

SEPTORIA LEAF SPOT

Septoria leaf spot, caused by *Septoria bataticola* Taub., is of little or no economic importance. It is widely distributed in the United States, and has been reported by Bruner (*17*) from Cuba and by Carpenter (*23*) from the Hawaiian Islands. Septoria leaf spot is characterized by small spots 2 to 5 millimeters in diameter with white centers scattered more or less indiscriminately over the upper surface

[6] Thümen, F. von. Mycotheca universalis. [Exsiccati] No. 598. 1876.

of the leaf. (Pl. 16, A.) The grayish white center in which is buried one or more pycnidia is surrounded by a brown border. The pycnidia are black and so minute as to be scarcely visible to the unaided eye. Usually only one pycnidium is present, though occasionally there may be two or more. According to Taubenhaus (*177*) the pycnidia vary in diameter from 70 to 130 μ. The spores are hyaline, 3 to 7 septate, curved, rarely straight, and measure 8.5 to 60 by 1.0 to 1.7 μ. (Fig. 16, C.)

Septoria bataticola occurs mostly on the mature leaves of sweet potatoes and makes its appearance in June or later, the date of appearance, however, depending on weather conditions and the geographical location. Septoria leaf spot has not been reported from any foreign country except Cuba. It is never serious enough to require remedial measures.

ALTERNARIA LEAF SPOT

A species of Alternaria is commonly found on the leaves of sweet potatoes, but it is difficult to say to what extent it is responsible for the dead spots in which it occurs. Attempts to infect leaves artificially with pure cultures of the organism have all given negative results. It is believed that the fungus is usually a secondary invader and gains entrance through wounds or tissues debilitated from the action of other fungi. On the other hand, it has been easy to isolate an Alternaria from dead spots of a certain type on sweet-potato leaves. Plate 16, D, shows a leaf with several characteristic Alternaria spots from which the fungus was isolated in pure culture. Infections of this type usually occur on the older leaves about the center of the hill. A number of leaves are usually attacked in a similar manner, indicating that it probably spreads from a primary infection. So far as could be determined, there was no injury to the leaves which would serve as a source of entrance for the fungus.

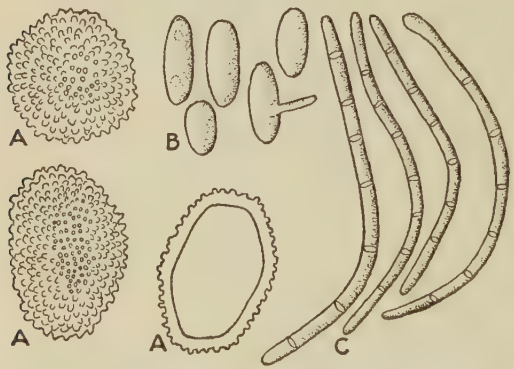


FIG. 16.—A, Uredospores of *Coleosporium ipomoeae*; B, conidia of *Monosporium uredinicolum*; C, conidia of *Septoria bataticola*. × 1,200

The well-known white-rust fungus, *Albugo ipomoeae-panduranae*, the pustules of which are restricted largely to the lower surface of the leaves, injures the tissue so that there is a loss of chlorophyll and a general debilitation of the leaf. On the upper surface of the leaf above such spots an Alternaria frequently gains a foothold, producing a brown spot of dead tissue with several concentric rings. (Pl. 16, D.) An Alternaria has also been isolated from other types of spots, as, for example, Phyllosticta spots and from spots presumably produced by sun scald. The evidence seems to indicate that the Alternaria enters wounds or lesions, and after it once gains a foothold it is able to continue its development and invade healthy tissue.

RUST

Rust (caused by *Coleosporium ipomoeae* (Schw.) Burr.) has been reported (*5*, *6*) on sweet potatoes in some of the West Indies, in Haiti (*60*), in Guatemala, and in Mississippi and Mexico. There is no record of its occurrence outside of the Western Hemisphere. It is probably common in the Tropics. Rust has also been reported on a number of species of Ipomoea in various parts of the United States (*5*), from New Jersey to Illinois and Kansas, southward to Florida, the West Indies, and Central America; also in South America. The type locality is North Carolina, where it is found on *Ipomoea triloba*.

Rust is not regarded as a serious disease of the sweet potato. According to Stevens (*167*), it is common but usually not destructive on sweet potatoes and other species of Ipomoea.

Only two stages (*5*) of this rust are known, the uredinial and the telial. These two stages are described as follows:

Uredinial stage.—Uredinia hypophyllous, widely scattered, or somewhat clustered, 0.25–1 millimeter across, early naked, orange-yellow fading to white, ruptured epidermis usually inconspicuous; urediniospores ellipsoid, 13–21 by 18–27 μ, more or less angular and irregular; wall thin, 1–1.5 μ, closely and noticeably verrucose. [Fig. 16, A.]

Telial stage.—Telia hypophyllous, widely scattered, often confluent, pulvinate, 0.5 millimeter or less across, deep reddish orange, fading to pale yellow; teliospores with wall swelling 20–40 μ above; contents orange-yellow fading to colorless, oblong, or slightly clavate, 19–23 by 60–80 μ, rounded or obtuse at both ends.

Although this organism has been identified from a number of different species of Ipomoea, the writers are of the opinion that the one occurring on *I. hederacea*, for example, is different, at least biologically, from the one reported on the sweet potato. *I. hederacea* occurs very commonly in sweet-potato fields in a number of States. It has been observed on numerous occasions with many rust pustules on the leaves of plants growing among sweet potatoes, but no infections of the sweet potato have ever been noted. In fact, the writers have never seen a Coleosporium on sweet potatoes in the United States, and it has never been reported to occur on them, except once in Mississippi. In view of the prevalence of many of the Ipomoeas on which it occurs in the regions where sweet potatoes are grown, it is surprising that it does not occur on the sweet potato. A study of the pathogenicity of this rust from different hosts might give indications that they are at least biologically different.

Coleosporium ipomoeae in Porto Rico, at least, is parasitized by *Monosporium uredinicolum* Stevens, and according to Stevens (*168*) each rust sorus at the time and place where he collected material was overgrown with this fungus. The hyaline mycelium was found covering the sori and growing in and around each rust spore.

Monosporium uredinicolum is described as follows:

Mycelium floccose, byssoid, forming white, moldy spots 1–2 millimeters in diameter over each rust sorus. Hyphae hyaline, septate, very sparsely dichotomously branched. Conidiophores indistinguishable from the mycelium, simple or sparsely dichotomously branched. Spores [fig. 16, B] acrogenous, solitary or rarely catenulate, 12–15 μ hyaline, continuous, cylindrical, obtuse at each end.

CERCOSPORA LEAF SPOT

In 1904 Zimmerman (*211*) described a species of Cercospora, *C. batatae* Zimm., occurring on sweet potato in Africa. Since then it has been reported from Brazil (*10*), China (*142*), Japan (*99*), and the Philippine Islands (*203*). Stevenson (*172*) reports the organism as causing irregular dark-brown leaf spots on *Ipomoea batatas* in Tanganyika, Indo China, Japan, Formosa, and the Philippine Islands. Bruner (*17*) mentions a *Cercospora* sp. on sweet-potato leaves in Cuba which may or may not be the same as the one described by Zimmerman and reported by other mycologists from parts of the the Eastern Hemisphere. *C. batatae*, according to Zimmerman, forms dark-brown, somewhat light-centered spots from 4 to 8 millimeters diameter. They are somewhat irregular in shape, being limited by the finer veins of the leaf. Zimmerman describes *C. batatae* as follows:

Die Konidienträger dieses Pilzes brechen in grosser Anzahl aus den Spaltöffnungen der Ober- und Unterseite des Blattes hervor; sie sind meist mit 2 Querwänden versehen, hellbräunlich, 35–45 mik. lang und 4–5 mik. breit. Die Konidien sind zylindrisch, am Vorderende etwas verschmälert, häufig gebogen, hyalin oder sehr hell bräunlich gelblich, mit 4–6 Querwänden, 60–100 mik. lang und 3–4 mik. breit.

FIG. 17.—Conidia of *Cercospora batatae*. × 1,200

According to Reinking (*143*) *Cercospora batatae* caused severe damage to sweet potatoes in southern China in 1919, producing spots nearly black extending through the entire leaf. Up to that time the disease was not known in the Philippine Islands, but since then Welles (*203*) reported a species of Cercospora, which he suspected to be *C. batatae*, on the leaves of sweet potatoes grown on the grounds of the Department of Agriculture, Los Banos, P. I., and in China (*204*). R. D. Rands has informed the writers that *C. batatae* caused considerable damage to sweet potatoes in Java, the disease being worst during the wet season, and as a consequence the crop is grown only to a very limited extent during the dry season. The disease was reported from Florida in 1923 (*18*), but it has never been reported from other States, indicating that it is likely to be more or less restricted to tropical or subtropical climates. It is also possible that the Cercospora occurring in Florida is not the same species as the one occurring in South Africa or in China, since a comparison of the fungus from Florida with the description of *C. batatae* reveals certain morphological differences. *C. batatae* is described as having spores 60 to 100 μ long, 3 to 4 μ wide, and 4 to 5 septations. The spores of the Cercospora from Florida on the other hand were 50 to 150 μ long, 4 to 5 μ wide, with 7 to 14 septations. (Fig. 17.)

The disease is not generally regarded as serious, but Welles (*204*), who noted considerable damage caused by it in one or two localities in

the Philippines, recommends spraying once in every two weeks with Bordeaux mixture, when the application of control measures is necessary.

SCLEROTINIA

A species of Sclerotinia has been reported only a few times to cause a disease of sweet potatoes. Poole, in New Jersey, found a Sclerotinia similar to *S. minor* Jagger, causing a damping off of several hundred sprouts of the Vineless Yellow Jersey in a seed bed in a greenhouse. The sprouts were very thick, so that a relatively high humidity would be maintained about the stems at the surface of the soil. Numerous small sclerotial bodies were found on the plants, but no apothecia. A Sclerotinia has been reported on sweet potatoes from Formosa (*160*).

MACROSPORIUM FOOT ROT

Taubenhaus (*184*) in 1925 described a disease of sweet potatoes and tomatoes due to *Macrosporium solani* E. and M. which causes deep dark lesions at the foot of the plant similar to the injury caused by Rhizoctonia and which in severe cases causes the plant to break over. Taubenhaus demonstrated by inoculation experiments that the organism isolated from either sweet potatoes or tomatoes would cause a similar disease of either crop. This disease is not prevalent on sweet potatoes, although it has been observed in Delaware and Texas by Taubenhaus. It is not unlikely that it may occur elsewhere.

OTHER FUNGI REPORTED

A Cylindrosporium, *C. bakeri* Syd., was reported by Weston (*206*) from the Philippine Islands. Ideta (*99*) reports *Helicobasidium mompa* N. Tanaka from Formosa, and Shirai (*160*) *Hypochnus centrifugus* (Lev.) Tul. and *H. sasakii* Shir. from the same country. A Macrophoma, *M. edulis* d'Almeida, was reported from the Azores by d'Almeida (*4*), and *Marasmius sacchari* Wak. from the West Indies by Nowell (*138*). An unknown species of Marasmius was reported from Barbados (*2*) and from New Zealand by Kirk (*105*). Two species of Meliola, *M. clarulata* Wint. (*166*) and *M. ipomoea* Earle (*170*), have been reported from Porto Rico.

Other fungi reported are *Schizophyllum commune* Fr. and *Sclerotium* sp. by Reinking from the Philippines (*143*), *Sclerotinia libertiana* Fuckel, and *Thielaviopsis ethaceticus* Went. from Formosa by Shirai (*160*), and *Vasculomyces xanthosoma* Ashby from Jamaica (*8*). Sawada (*152*) reports the following fungi occurring on sweet potatoes in Formosa: *Helicobasidium mompa* Tanaka, *Hypochnus centrifugus* (Lev.) Tul., *H. sasakii* Shir., *Sclerotinia libertiana* Fuckel. Nothing is said as to the damage caused by these organisms. In South Africa Doidge (*37*) lists the following diseases: Brack injury, scab (*Actinomyces* sp.), and two storage rots due to *Fusarium* sp. and *Rhizopus nigricans*, in Cape Province; leaf spot (*Cerospora* sp.) in Natal; and the phanerogamic root parasite, *Striga orobanchoides*, which destroyed large patches of plants in the Transvaal. A wet mold (*Chenophora* sp.) was reported from Florida by Burger (*18*). Stevenson (*172*) reported *Ramularia batatae* Rac. as causing angular dark-brown to brown leaf spots on *Ipomoea batatas* in Java.

SLIME MOLDS

Slime molds are common on stems, petioles, and leaves of sweet-potato plants in the hotbed (pl. 17, B), particularly in beds under glass where the foliage is very dense. Slime molds have been collected on numerous occasions and reported by other investigators from various parts of the United States (*134*, *183*, *185*). Slime molds can not be regarded as parasitic on sweet potatoes. They may appear in spots of various sizes quite suddenly in the hotbed if the temperature and relative humidity are high. The leaves on which they grow are sometimes dwarfed and slightly distorted, probably as a result of the interference in the normal photosynthetic activity of the plant. They often develop so abundantly that the entire surface of the leaf and petiole is covered.

These slime molds produce a cottony, white, yellow to purplish slimy coating on any of the aboveground parts of the plant. In a short time the jellylike mass changes to a brownish powder, consisting largely of spores, which may be liberated and carried about by the wind or other mechanical means.

Two different slime molds have been reported on sweet potatoes, *Fuligo violacea* Pers. and *Physarum plumbeum* Fries. *F. violacea* is the organism most frequently found, although *P. plumbeum* has been reported from Alabama, Arkansas, and Delaware.

MAJOR STORAGE DISEASES

The quantity of sweet potatoes stored each year can be only roughly estimated. Approximately 84,000,000 bushels were produced in 1926, and it is probable that about 30,000,000 to 40,000,000 bushels were placed in storage, the remainder having been sent to the market when dug or consumed locally. It is likewise difficult to estimate with any accuracy the quantity of stored potatoes lost each season due to the several storage-rot organisms. The losses in recent years are doubtless less than formerly, owing to the fact that improved methods of handling and storage have been practiced. The installation of modern storehouses throughout the country and the increased knowledge of how to operate them have resulted in a great reduction in the amount of loss. In spite of recent progress in storage-house construction and operation, the annual loss probably reaches 5 to 40 per cent of the crop stored. Manns (*118*) estimated the loss in storage to be 20 per cent in Delaware, while Neal (*126*) and Elliott (*48*) calculated it to be 25 and 30 per cent in Mississippi and Arkansas, respectively. If the losses in these States are representative of those in other States, they amount to many millions of dollars annually. Very little literature is published regarding the storage of sweet potatoes in foreign countries or the losses there. Sweet potatoes are stored to some extent in Japan, but very little information is available regarding the loss sustained in storage in that country.

In the United States three general methods are employed in the storage of sweet potatoes—in banks, in cellars, and in specially constructed houses. The bank method, which has been long in use, consists in heaping the potatoes in a pile out of doors and covering them first with pine needles, straw, cornstalks, or similar material,

and then with 4 to 6 inches or more of dirt. The covering must be sufficient to protect the potatoes against freezing. In some cases a box about 6 inches square with numerous holes bored through the sides is placed on end in the center of the pile to serve as a means of ventilation. This device, however, is not always used.

Instead of storing in banks, many growers have used, and still use, a cellar. The cellars are not always well ventilated, and consequently heavy losses often result. With the expansion of the industry and the increase in knowledge of the principles underlying the storage of sweet potatoes there developed the modern storage house in which these principles could be applied. Plans for the construction of an up-to-date storage house can be obtained from the Bureau of Public Roads, United States Department of Agriculture, Washington, D. C.

Although any one of the above methods may be used for storing sweet potatoes, the storage house is the most uniformly successful. Even in the dirt bank, sweet potatoes frequently keep very well. The success with which they are kept in banks, however, depends very much upon the condition of the potatoes when they are harvested. Barre (*13*), as a result of experimental evidence, concluded that it is safe to store in banks in South Carolina, provided the potatoes are free from disease when stored and the banks are properly made. However, in spite of the occasional success in bank storage, the losses one season with another have been so large that the introduction of the modern storage house has been inevitable.

For convenience in presentation the diseases of sweet potatoes in storage will be considered separately, the more important ones being taken up first.

About 50 species of fungi have been isolated from decayed sweet potatoes. A considerable number of them were found to be saprophytes that gained a foothold in lesions made by some of the parasites. In fact, it has been found that certain saprophytes frequently gain a foothold after the parasite has died. Frequently several different species of fungi can be isolated from the same decayed potato, which suggests that the only sure way of determining the parasite is to make isolations and then inoculate with the different fungi from pure cultures. This has been done with a large number of organisms, and 17 were found capable of causing decay in sweet potatoes (*89*) under suitable conditions. To these a number of other species can now be added. Only a few species are important as producers of storage rot, the others being found only under conditions especially favorable for their development. It is interesting to note that some of these organisms that are rarely met with under natural conditions cause decay only when they are removed from the competition of other fungi.

SOFT ROT AND RING ROT

Soft rot and ring rot are caused by a group of closely related fungi belonging to the genus Rhizopus and especially to the species *nigricans*. The most common species and the two causing most of the soft rot in storage are *Rhizopus nigricans* Ehrb. and *R. tritici* Saito (*108*), the former at temperatures between 6° and 20° C, and the latter at 30° and above, the two overlapping between 20° and

30°. Seven other species of Rhizopus—namely, *R. artocarpi* Racib., *R. delemar* (Boid.) Wehmer and Hanzawa, *R. maydis* Bruderl., *R. nodosus* Namysl., *R. oryzae* Went. and Pr. Geerligs, *R. reflexus* Bainier, and *R. arrhizus* Fischer—were found to cause soft rot also, but they were unable to compete with *R. nigricans* at temperatures of 12° to 18°. Two other species, *R. chinensis* Saito and *R. microsporus* Van Tieghem, were studied and found incapable of decaying sweet potatoes (*90*). The decay caused by the different species was identical so far as macroscopic appearances were concerned. Soft rot is the most common and destructive of the storage-rot diseases. *R. nigricans* occurs in all storage houses and when conditions are favorable causes enormous losses. The so-called soft rot begins at one of the ends of the potato, occasionally elsewhere, and progresses rapidly through the healthy tissue. Only four to six days under favorable conditions are required to complete the destruction of the entire potato, experimental data having shown that after decay has once started it will continue, though it is slightly retarded in an atmosphere almost entirely free from moisture.

The potatoes are at first rendered very soft and stringy, water often dripping out of the potato when it is broken open. Soft rot has a characteristic mild yeast odor at first, followed by a wild-rose to rose-geranium odor later. At the outset the color of the tissue is not changed, but later it becomes cinnamon to chocolate brown. If the skin of a decayed potato is ruptured, the sporangiophores and sporangia develop in great numbers from the exposed tissue. (Pl. 18, A.) On the escape of moisture the potato dries up, finally becoming dry and mummified. Observed in this stage it is often classed as dry rot.

Soft rot often sets in soon after the potatoes are put in storage and continues more or less throughout the entire storage period, depending largely upon the condition of the potatoes when they are harvested and on the management of the storage house. Soft rot is largely a storage trouble, though it is occasionally found in the field at digging time in wet soils, especially those containing considerable organic matter.

Ring rot differs from soft rot only in that the infection occurs at one or more places between the two ends. It progresses around the potato, forming a ring or collar by the drying out and subsequent shrinking away of the rotted tissue. The extent of the rot varies, being in some cases 1 or 2 inches in width, and it may extend one-half inch in depth or entirely through the potato. (Pl. 18, B.) The diseased tissue may dry up after the ring has been completed and the rot make no further progress, or it may advance toward the two ends and finally complete the destruction of the entire potato. As many as three such rings have been seen on one potato. Ring rot was first said to be caused by *Nectria ipomoeae* (*65*), but was later shown by Taubenhaus (*177*) to be caused by *Rhizopus nigricans*, the fungus usually responsible for soft rot under storage conditions.

In view of the general prevalence and wide distribution of *Rhizopus nigricans* it is doubtful whether dissemination over any great distance is ever necessary to insure infection. Even though the storage house has been thoroughly cleaned and disinfected, many of the spores doubtless are carried into the house with the potatoes,

where they remain dormant until conditions are favorable for germination. If infection results, as it may in even the best-kept houses, the spores may be formed in great abundance on the surface of the decayed potatoes, from which they are borne to others. There are, therefore, many chances for infection. In spite of the fact that this organism causes enormous losses in storage houses, it has been very difficult to obtain infection by inoculation. It has been demonstrated that infection will not take place through the unbroken skin, but that a wound of some sort must be provided. As a matter of fact, it has been demonstrated that infection will not always result when the spores and hyphae are smeared on a fresh wound. Rhizopus produces an enzyme pectinase (*86*, *87*) which dissolves the middle lamellae and thereby aids in infection. Infection, therefore, results more readily if the spores are first germinated in some nutrient medium, such as sweet-potato decoction, in which the enzyme is abundantly produced.

Investigations have shown that infection rarely takes place except through fresh wounds. Studies by Lauritzen and Harter (*110*) have demonstrated a relationship existing between the temperature and humidity at which the potatoes are held and of their susceptibility to infection: i. e., if wounded, the potato is liable to infection, provided certain other conditions, such as a suitable temperature and humidity, are present. The optimum humidity for infection is not necessarily the highest possible humidity. At 23° C. a larger percentage of infection occurred at a relative humidity of 75 to 84 per cent (the optimum) than at 93 to 99 per cent. At this temperature the percentage of infections decreased as the relative humidity was raised or lowered. The conclusions to be drawn from these results are (1) that fresh wounding is necessary to infection and (2) that infection of freshly wounded potatoes is not so apt to occur at relative humidities of 93 to 99 per cent as at humidities somewhat lower; as, for example, 75 to 84 per cent.

According to Lauritzen and Harter (*109*), the time required for infection of wounded sweet potatoes by Rhizopus varies from five to seven days at 9° C. to 43 hours and less at from 18° to 32°. The time varies according to the extent of wounding. The six species studied by them were roughly placed in high (*R. tritici*, *R. oryzae*, *R. maydis*) and low (*R. nigricans*, *R. reflexus*, *R. artocarpi*) temperature groups, according to their temperature responses. The optimum for the high-temperature group varied from 32° to 35°, the maximum was about 42°, and the minimum 4.5° to 9°. The optimum for the low-temperature group varied from 18.5° to 24°, the maximum from 30° to 34.5°, and the minimum from 3.4° to 12°. The extreme temperatures over which infection would take place were between 3.4° and 42°.

Investigations made by the writers have shown that all the varieties studied are more or less susceptible to infection by the different species (*88*) of Rhizopus. With respect to the readiness with which they decay, it was found that the varieties can be divided into three groups—those that are very susceptible (Gold Skin, Yellow Jersey, Belmont, Red Brazil, Haiti, Yellow Yam, and Dooley), those that are quite resistant (Nancy Hall and Southern Queen), and

those that are intermediate between the first two (Porto Rico, Big-Stem Jersey, Triumph, Pierson, Florida, and Dahomey). It seems to be quite generally agreed that the Southern Queen keeps well in storage, but not all the investigators have agreed with respect to the Nancy Hall. Some detailed work carried out by Lauritzen, in which the extent of decay at the end of a certain period was actually measured, shows that Nancy Hall is not so resistant as the results of investigations by the writers seem to indicate.

BLACK ROT

Black rot, caused by *Ceratostomella fimbriata* (E. and Hals.) Ell., is both a field and a storage disease. The characteristic symptoms of this disease in the field have been detailed elsewhere (pp. 21 and 22) and will be reviewed here only so far as it is necessary to show its connection with the decay in storage. The morphology of the causal organism, its distribution, mode of dissemination, and prevalence are given under "Field diseases" (p. 24). Black rot frequently causes very large losses in storage houses, and especially if the disease occurs in the seed bed and in the field.

If black-rotted potatoes are bedded, the slips produced from them are likely to have black rot. If such plants are set in the field the potatoes produced may have black-rot lesions on them, and it is on these infected potatoes that the disease is carried from the field into the storage house. A large number of black-rot infected potatoes is observed in the field only in case of a severe epidemic. However, there may be a large number of infections too small to be seen with the unaided eye which gradually enlarge in the storage house and at the end of four to six weeks attain a diameter of an inch or more. The rapidity with which the spots enlarge depends upon the condition of the storage house, largely with respect to temperature and humidity. Black rot develops very slowly at a temperature below 10° and above 35° C., the optimum, maximum, and minimum temperatures (*106*) for infection and the development of the disease being 23° to 27°, 34.5° to 36°, and 9° to 10°, respectively. The rate of development of an infection is very slow between 6° and 14°, but it increases very rapidly above 14° until it reaches a maximum between 23° and 27°. The number of infections is governed by the temperature which prevails during the early stages of infection (11 days). Plate 5, A and B, shows two photographs of an infected potato before and after it had been maintained for two months at a temperature of 10° to 13°.

The black-rot fungus may be disseminated from potato to potato in the storage house in several ways. This organism, as previously pointed out, develops perithecia in which are formed myriads of hyaline one-celled spores, which exude from the end of the fimbriated beak in a slimy mass and thence may be borne on the bodies of insects, by rodents, or similar agencies to other potatoes and thus start new infections. It is probable that the spores may be scattered about the storage house by air currents, by the settling of the potatoes in the bins, and by other means, such as handling the potatoes in preparing them for the market.

JAVA BLACK ROT

Java black rot, caused by *Diplodia tubericola* (E. and E.) Taub., is very widely distributed, and the total loss caused by it is large. Sweet potatoes infected with this organism have been received from Cuba, Isle of Pines, the Philippine Islands, Japan, Porto Rico, South America, and other countries. It has been collected in every part of the United States where sweet potatoes are grown. The disease causes great loss in the Tropics and in the southern part of the United States. Sarmiento (*151*) has shown that in the Philippines insects may be partly responsible for the distribution of Diplodia.

Java black rot was first observed in 1896 by Clendenin (*28*) on sweet potatoes sent to the Louisiana Agricultural Experiment Station from Java. From what is known at the present time, it is likely that this disease was common in this country long before that time; probably it has been common as long as sweet potatoes have been cultivated, since it has been shown that various species of Diplodia occurring on different hosts will infect sweet potatoes.

Harter (*73*) showed that *Diplodia tubericola* from the dasheen (*Colocasia esculenta*), *D. gossypina* Cke., *D. maclurae* Speg., and *Diplodia* sp. from mango (*Mangifera indica*) will produce a rot of sweet potatoes identical with that produced by *D. tubericola* isolated from sweet potatoes; it has also been found (*179*) that *D. natalensis* Ev. and *Lasiodiplodia nigra* Appel and Laub. will cause a rot of sweet potatoes similar to the rot caused by *D. tubericola*. Further evidence of the cosmopolitan nature of these organisms has been published by Meier (*122*), who found that *D. tubericola* from the sweet potato would cause stem-end rot of the watermelon (*Citrullus vulgaris*).

Diplodia tubericola rots sweet potatoes very slowly. There is little or no evidence of infection under laboratory conditions for a week or 10 days following inoculation, and usually four to eight weeks are required to decay a potato entirely. (Pl. 19, A.) The pycnidia, which develop in considerable numbers, generally appear after about one month on the part of the potato first decayed. They are externally coal black, crowded closely together or confluent (fig. 18), and form minute, domelike elevations on the surface. Unlike many of the fungi of this group, some of the pycnidia are completely buried, the spores escaping only after maceration or disintegration of the host tissue.

In the early stages of decay the tissue is brown in color, but it later becomes coal black and hard. (Pl. 19, A.) Concomitantly with the escape of water, the potato shrinks, eventually becoming mummified.

The spores may be of three types, and sometimes all three are found in the same pycnidium. In the young pycnidium they are usually hyaline and one-celled (fig. 19, C), and occasionally this is the only type found. Later the hyaline spores may turn dark (fig. 19, B) and they may (fig. 19, D) or may not become septate. In the old mummied potato the 2-celled dark spores predominate, but are intermixed with a few 1-celled dark and a few 1-celled hyaline spores.

Numerous inoculation experiments have demonstrated the pathogenicity of this organism (*89*).

The pycnidia are more or less globose (fig. 18), 250 to 305 μ in diameter; the stromatic mass is about 1 millimeter in diameter. The conidia are elliptic, not constricted, 18 to 20 μ by 11 to 14 μ. The paraphyses are 45 to 65 μ in length. (Fig. 19, A.)

DRY ROT

Dry rot is caused by a fungus known as *Diaporthe batatatis* (E. and H.) Harter and Field, reported by Halsted (*61*) in 1890, who attributed it to *Phoma batatae* E. and H. Its ascigerous stage, to which the name *D. batatatis* was given, was later obtained by Harter and Field (*79*). The conidial stage of the fungus is the only one found on the potatoes from the storage houses or from field material. Dry rot has been reported to occur in North Carolina, Texas, New Jersey, Virginia, Mississippi, Alabama, and Indiana, so that it can safely be said to have a wide distribution. An organism isolated

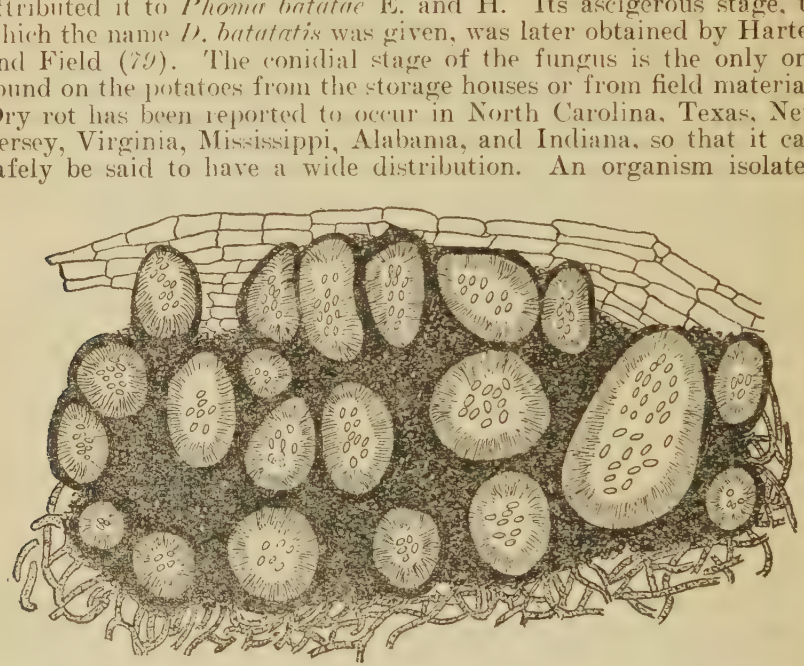
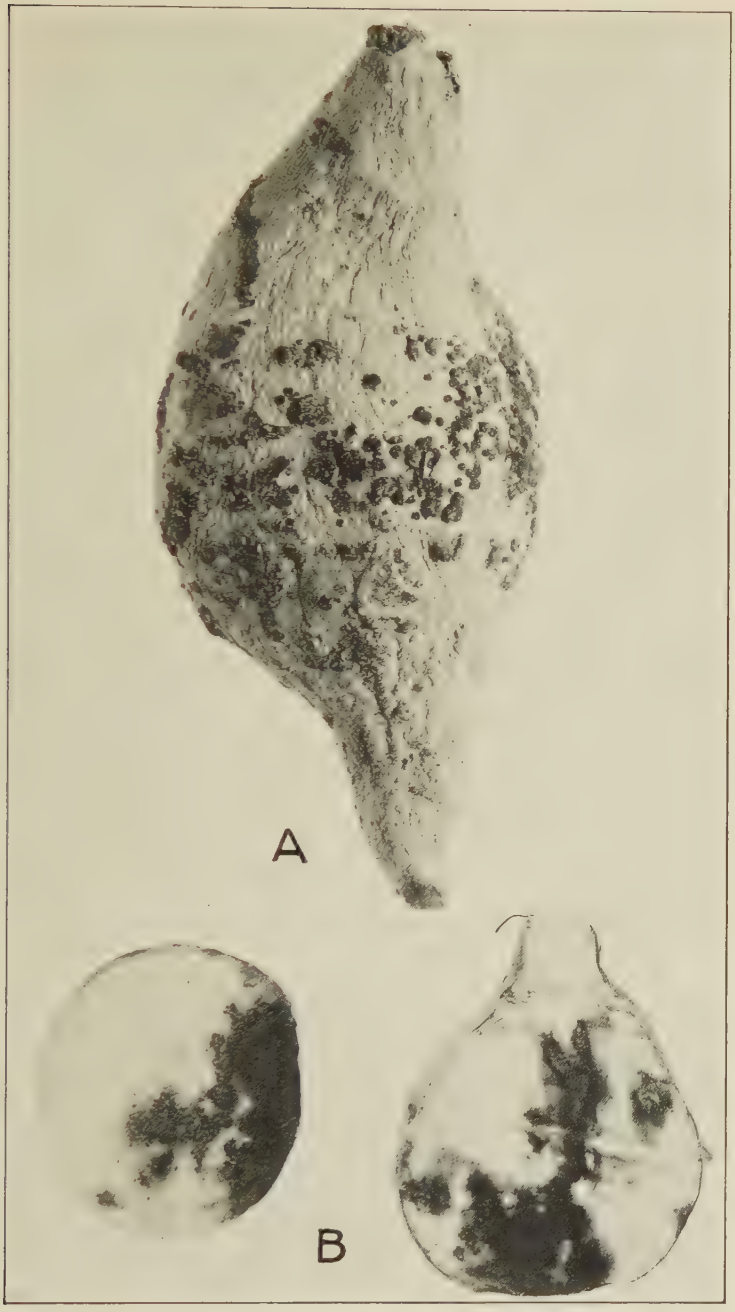


FIG. 18.—Pycnidia of *Diplodia tubericola* embedded at different depths of the tissue of the host. × 97

from sweet potatoes from the Isle of Pines when inoculated into sweet potatoes in the United States produced the characteristic symptoms of the disease. This strain, while identical morphologically, is a more vigorous parasite than any isolated from material collected in the United States.

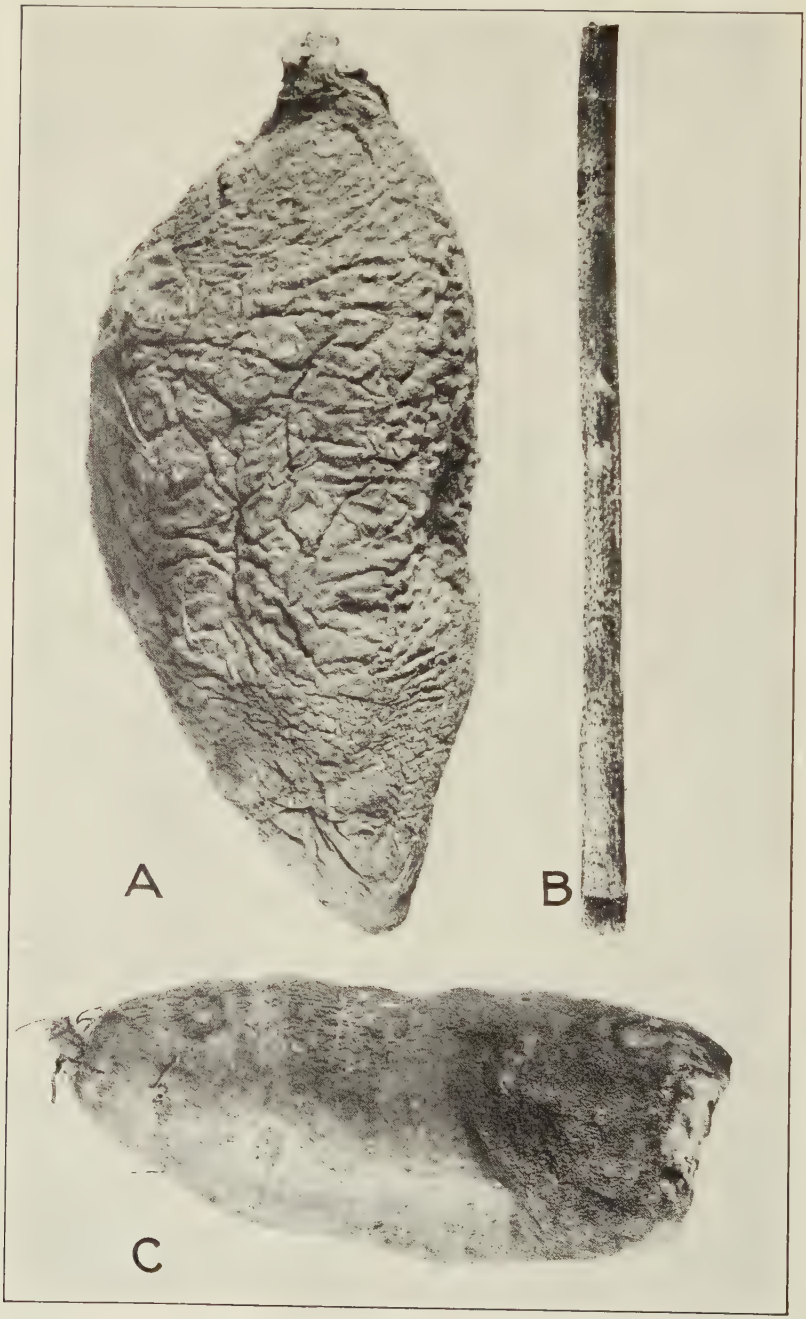
The total loss from this disease is relatively small, being greater in storage than in the field. It is occasionally found on the slips in the hotbed and on the vines (Pl. 20, B) in the field. Like many other fungi it will grow as a saprophyte and for this reason is found as a secondary invader.

Inoculation experiments have shown that it is capable of causing decay in storage, requiring four to eight weeks to rot a potato entirely. Infected potatoes become much shrunken and wrinkled (pl. 20, A) and finally mummified. The surface, beneath which the tissue is carbonaceous to coal black, is covered with small elevations a millimeter or so in diameter, lying close together, in which the pycnidia are embedded. (Pl. 20, C.)



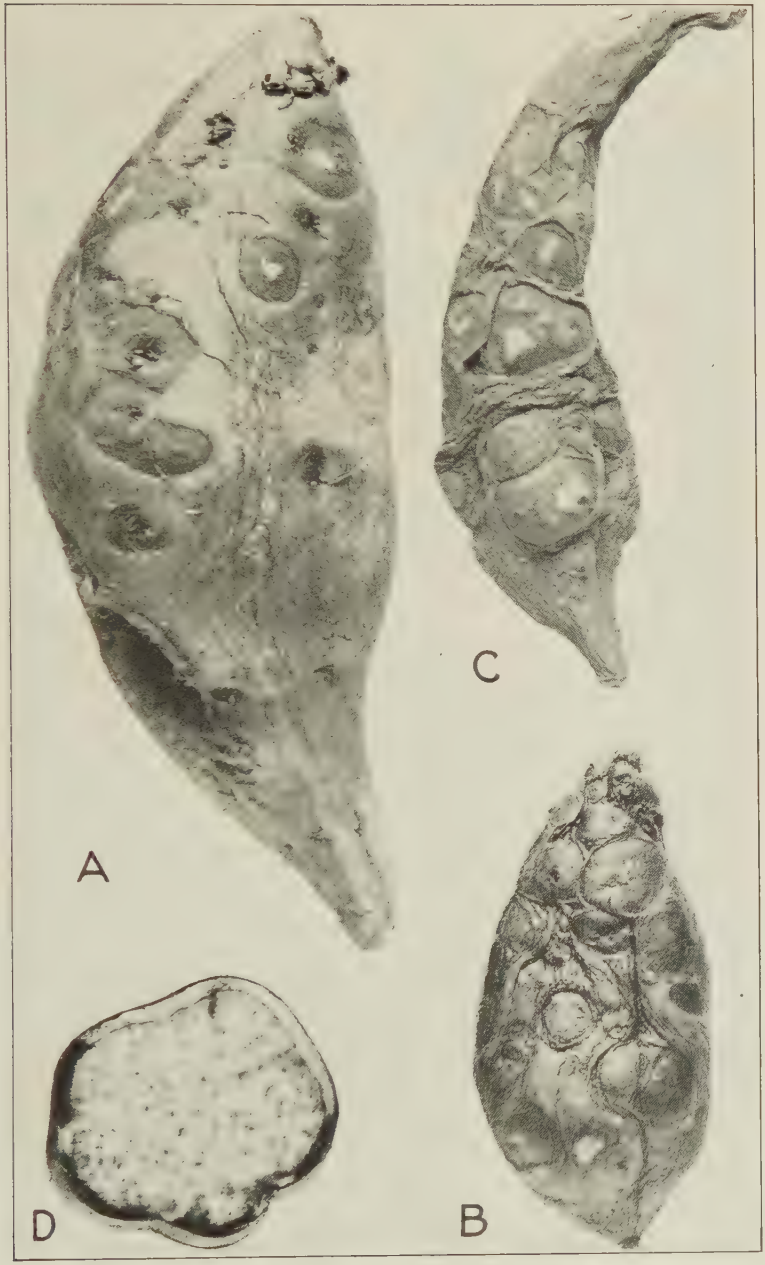
TWO SERIOUS STORAGE ROTS

A.—Java black rot (*Diplodia tubericola*). Specimen completely decayed, dry, and hard. Note the black cushions on the surface, in which numerous pycnidia are embedded. B.—Charcoal rot (*Sclerotium bataticola*). The tissue is rendered black by the numerous sclerotial bodies that form soon after decay sets in



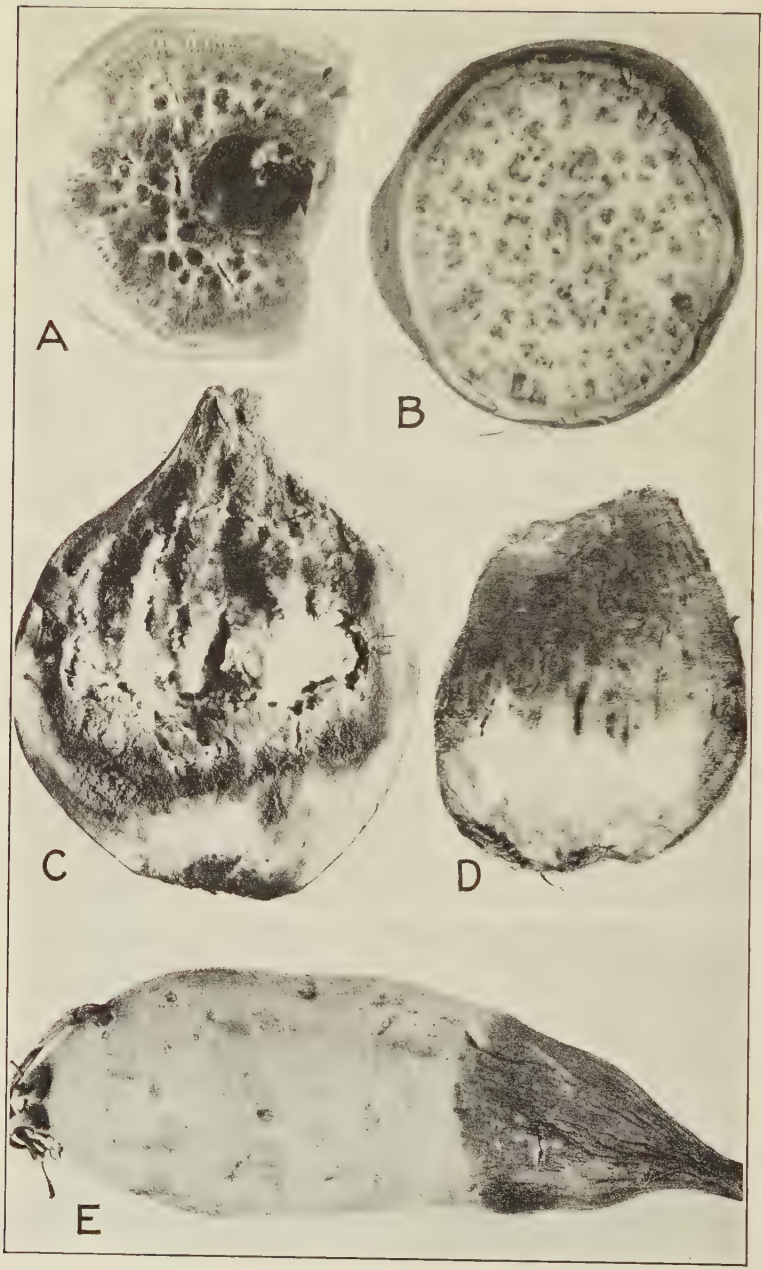
DRY ROT (DIAPORTHE BATATATIS)

A.—A sweet potato completely decayed. The potato eventually dries, becoming hard and mummified. B.—Infection on the vine, showing the presence of numerous pycnidia. C.—Potato only partially decayed. Numerous pycnidia may be seen on the surface of the decayed portion



SURFACE ROT (FUSARIUM OXYSPORUM)

A.—An early stage of the disease in which the injury is restricted to small areas on the surface of the root. B.—Later stage with large spots and considerable shriveling. C.—Mummified potato, an extreme case of surface rot. D.—Cross section of root, showing the depth to which the organism penetrates



MINOR STORAGE ROTS OF SWEET POTATOES

A.—*Epicoccum* sp. This organism destroys the potato slowly, producing a firm rot, at first yellowish, then reddish brown. B.—*Mucor racemosus*. This organism causes a rather slow rot at low temperatures. The potato is rendered wet but spongy to firm. C.—*Botrytis cinerea*. This fungus produces a grayish, soft, somewhat watery rot with a slightly starchy odor. D.—*Alternaria* sp. A firm moist rot is produced. At first the tissue is turned brown, and then it gradually darkens. E.—End rot, probably caused by *Fusarium oxysporum*. A firm dry rot is produced. The tissue becomes brown and somewhat powdery

The pycnida at first covered by the epidermis but eventually breaking forth, are separate, occasionally confluent, and more or less globose, measuring 60 to 130 by 60 to 110 μ and having a short neck. Only on the roots are the pycnidia embedded in a stroma.

The conidia (fig. 20, C) are oblong to fusoid, 6 to 8 by 3 to 5 μ, continuous, hyaline, usually two guttulate; sometimes, however, with three oil droplets. They are borne on long hyaline, simple, continuous filiform conidiophores, which are either straight or curved. In the same pycnidium with the conidia there may or may not be found another type of spore, the so-called stylospores. These are filiform, hook-shaped bodies, sometimes curved or rarely straight, 16 to 30 μ in length, and continuous. (Fig. 20, A.) Stylospores and conidia may be found alone or together in the same pycnidium.

The perithecia are formed in a Valsalike stroma, which is ashy gray within and carbonaceous without, with many exserted subcylindrical beaks from 0.5 to 3 millimeters in length. They are subglobose, 120 to 370 μ in diameter. A tangential section shows the perithecia formed in the same stroma with the pycnidia, frequently in a ring inside but separate from the pycnidial chamber.

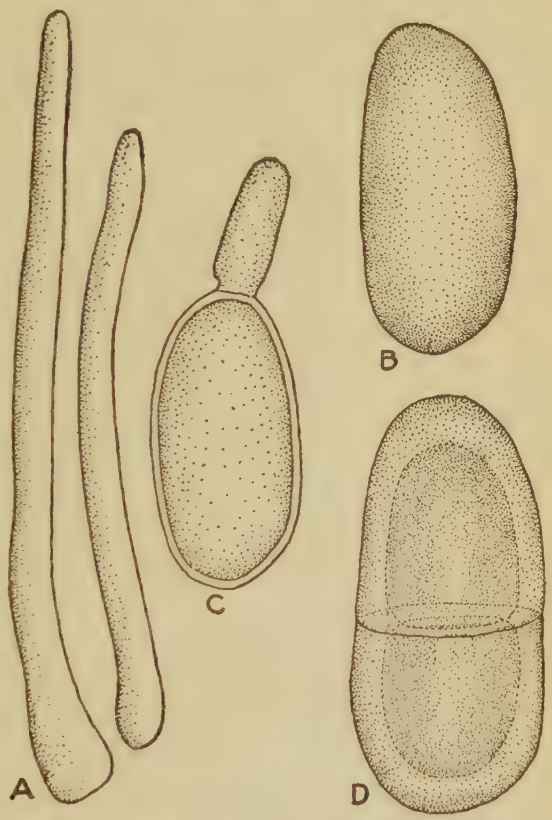


FIG. 19.—*Diplodia tubericola:* A, Paraphyses; B, one-celled brown conidium; C, one-celled hyaline conidium germinating; D, two-celled brown conidium. × 1,580

The asci are clavate to cylindrical in shape, sessile, eight spored, 23 to 38 by 7 to 12 μ. (Fig. 20, B.) The spores are subelliptical, obtuse at both ends, hyaline, one-septate, slightly constricted at the center, two to four guttulate, and 8 to 12 by 4 to 6 μ. No paraphyses are present.

The belief that the ascigerous stage represents the perfect stage in the life history of the dry-rot fungus is based on the following facts: (1) The ascigerous stage and the pycnidial stage were both derived from the same original isolation; (2) a conidial stage was developed from a single ascus that was identical with the original isolation; (3) infection was obtained through inoculation from the ascospore strain.

FOOT ROT

Foot rot, caused by *Plenodomus destruens* Harter, has been discussed primarily as a hotbed and field disease (pp. 27 to 33). It was previously pointed out that under field conditions the causal organism grows from the stem into the roots. (See pl. 7, B.) Sweet potatoes only slightly infected may, and frequently do, find their way into the storage house, where the fungus develops still further. Slightly decayed roots may be used for seed and the fungus grow from them to the sprouts and be carried by them to the field. Spores are abundantly produced, and some of them may be lodged in wounds or lesions made by rough handling. In view of this fact, potatoes that otherwise might appear to be sound may readily carry the fungus to the hotbed, where, upon the addition of moisture, infections may occur. Experiments have shown that bruised potatoes taken from a storage house where foot rot was present developed numerous pycnidia on their surfaces when they were held in a moist chamber for a few days.

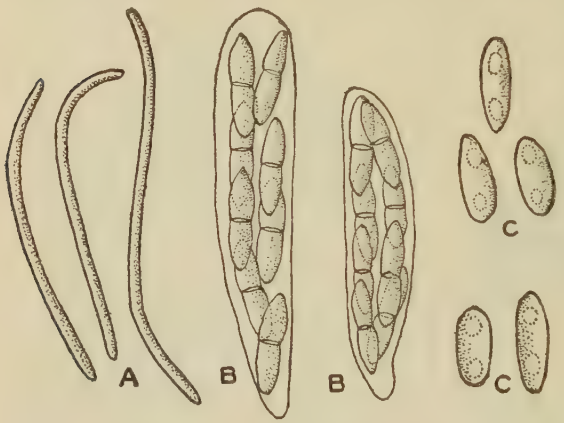


FIG. 20.—*Diaporthe batatatis:* A, Stylospores; B, asci; C, conidia. × 1,335

Plenodomus destruens slowly produces a relatively spongy brown-colored rot, two to three weeks being required to destroy completely an average-sized potato. Upon the escape of moisture the potato becomes dry, shrunken, hard, and finally brittle.

The parasitism of *Plenodomus destruens* has been demonstrated by numerous inoculations of both plants and potatoes.

CHARCOAL ROT

Charcoal rot (pl. 19, B) caused by the sterile fungus, *Sclerotium bataticola* Taub., has been collected in all parts of the United States, and sweet potatoes decayed by it have been received from Japan and other foreign countries.

Sclerotium bataticola was originally thought by Halsted (*61*) to be a stage in the life history of *Ceratostomella fimbriata*, but after a more thorough study of the fungus by Halsted and Fairchild (*67*) it was evident that they entertained some doubt of its connection with the black-rot fungus. Later investigations showed that *S. bataticola* was in no way connected with *C. fimbriata* (*176*).

Sclerotium bataticola is a slow-growing storage-rot fungus requiring about three to six weeks to rot a potato completely under moist-chamber conditions at laboratory temperature (18° to 24° C.). The decayed tissue first becomes chocolate to cinnamon brown, which later

changes to a dark reddish brown color. As soon as the sclerotia begin to form, the host tissue becomes black or charcoallike in appearance. Two distinct zones differing in color may often be distinguished at the same time in a single potato. The black zone, usually the outer one, contains the sclerotial bodies. Adjacent to this is a dark reddish brown area. The potato in the early stages of decay is spongy, but on the escape of moisture it gradually becomes hard and mummified. If the epidermis is broken, the black sclerotial bodies may be seen in large numbers buried among the cells throughout the potato.

Inoculation experiments (*89*) have demonstrated the parasitism of this organism. The coal-black sclerotia, made up of anastomosed black hyphae, vary in shape from spherical to oval, oblong, and even forked, and range in size from 22.4 to 25 by 32 to 152 μ. They are developed in great numbers throughout the potato and can be readily exposed by peeling away the epidermis.

Although *S. bataticola* is primarily a storage disease it may sometimes be found on the stem near to or just below the soil line on plants growing under field conditions, where it causes almost charcoal-black discolored lesions in the cortex. These lesions may enlarge and by harvest time may have extended from the initial point of infection to the roots. It is probable that many of the infections found on sweet potatoes in storage had their origin in the field.

SCURF

Scurf, caused by the fungus *Monilochaetes infuscans* Hals., like black rot and foot rot, is both a field (pp. 36 to 40) and a storage disease. Scurf is present in the United States wherever sweet potatoes are grown, forming rusty brown patches over the surface on all the underground parts of the plant. These discolored patches are often so numerous as to cause an almost continuous scurfy covering over all or nearly all of the sweet potato. The causal fungus penetrates only through the skin, so that it can not be said to cause a storage rot in the same sense as soft rot and black rot. Water escapes from badly scurfed potatoes, so that they become spongy and finally dry and hard, the loss being due largely to shrinkage. If scurfy potatoes are used for seed, however, they will produce scurfy plants on which the fungus is carried to the field. During the growing period in the field the fungus grows down or the spores are washed down the stem of the plant to the roots, on which it is carried to the storage house. The attached end of the potatoes is usually worst affected. Field infections of plants also take place. The disease is most severe in heavy black soils and in those containing a considerable quantity of organic matter. Sweet potatoes grown on soil to which stable manure has been added or where green-manure crops have been grown are frequently badly marked with scurf.

In severe cases of scurf the skin of the sweet potato in storage may crack (see pl. 10, D), permitting the water to escape. Scurf does not affect the edible quality of the potato, although its market value is considerably reduced (*30*). In spite of the fact that scurf bears very little resemblance to black rot, consumers are not always able to distinguish between them.

SURFACE ROT

Surface rot is caused by the fungus *Fusarium oxysporum* Schlecht. It is widely distributed, having been collected on specimens received from all parts of the United States. It attacks all varieties of potatoes more or less, though the Jersey types appear to be most susceptible. Observations indicate that greater losses are caused to the Big-Stem Jersey, year after year, than to any other variety studied or upon which observations have been made.

The early stage of surface rot is characterized by the nearly circular to roundish, somewhat sunken spots (pl. 21, A), the spots varying in number and differing in size according to the length of time that has elapsed since the infection started. They usually do not exceed three-fourths of an inch in diameter. The rot is shallow, seldom penetrating below the fibrovascular ring. (Pl. 21, D.) Later there is some shrinkage of the potato (pl. 21, B), especially at the margin of the spots and between them, if they are close together. Finally the potato becomes dry and mummified (pl. 21, C). Surface rot has some characteristics in common with black rot, but differs from it in several essential details. In the former the spots are smaller and grayish brown, while in the latter they may attain a diameter of 2 inches or more and are more nearly black. Surface-rot spots differ from bruises in that they are more regular in shape and size, being usually circular or nearly so, while spots caused by bruises may be irregular in shape and variable in size.

The results of investigations made by the writers (*84*) indicate that infection takes place at about digging time or early in the storage period when small decayed spots can frequently be found at the base of the small rootlets, especially if the ground is somewhat wet. These infected areas slowly enlarge in storage and become more or less conspicuous at the end of six to eight weeks. The disease is worst if the potatoes are dug during a wet time or when the ground is quite moist.

If the storage house is kept rather warm and dry the moisture escapes from the sweet potato through the surface-rot lesions, the potato eventually becoming hard and mummified.

The loss from surface rot is frequently very large, often exceeding that of any of the other storage diseases. Occasionally most of the potatoes in the house are so badly marked and shrunken that they have no market value. Recently Lauritzen (*107*) has developed by selection a strain of Yellow Jersey very little subject to injury from surface rot. It was noticed that those potatoes with a dark-yellow skin were not at all or were very little injured. A careful comparison of the dark-skinned potatoes with the light-skinned ones under conditions suitable for the development of surface rot showed that the former remained practically free, while the latter were badly diseased. Potatoes affected with surface rot shrink badly in storage.

Many inoculation experiments have demonstrated that *Fusarium oxysporum* is the cause of surface rot. Furthermore, as it has been shown that the stem-rot organisms do not cause surface rot, there is no danger of introducing or increasing stem rot by bedding potatoes affected with surface rot.

MINOR STORAGE DISEASES

The following group of storage rots caused by fungi are occasionally found. Under proper conditions these fungi will decay sweet potatoes. That they have not been more frequently encountered may be due to the fact that they are for the most part slow-growing forms and are therefore probably crowded out by the organisms that develop much more rapidly; as, for example, the black-rot fungus. Furthermore, some of them are conspicuously low-temperature forms and will not decay sweet potatoes, except at temperatures lower than those at which sweet potatoes are stored. Sweet potatoes are decayed by some of the organisms causing minor storage rots only if the competition of the more vigorous parasites is removed, or if the host is maintained at a low temperature or at a temperature congenial to the growth of the fungus, or when both these factors are operative.

As no common names are known for these rots, they will be considered under the names of the causal organisms.

MUCOR RACEMOSUS

Mucor racemosus Fes. seems to be capable of decaying sweet potatoes only at low temperatures (*89*). If the potatoes are held for several weeks at temperatures a little above freezing; that is, 2° to 5° C., a soft rot may begin at wounds or dead rootlets. Under such conditions the decay progresses slowly so that four or more weeks are required to produce somewhat sunken, nearly round, and often somewhat zonated spots of an inch or more in diameter. The center of each spot is usually somewhat bluish in color, because a Penicillium soon gains a foothold, living as a saprophyte on the decayed tissue. Both these fungi are frequently obtained in culture, unless the planting is made from the margin of the sound tissue, in which case Mucor alone is obtained.

M. racemosus produces a rot similar to that produced by *Rhizopus nigricans*, but much slower. The tissue, which has a distinctly starchy odor, is rendered a gray color in spots, as shown in cross section. (Pl. 22, B.) It is somewhat wet, spongy to firm, and fibrous and stringy when pulled apart or broken open.

A number of inoculation experiments have shown that *M. racemosus* is a parasite, that it will decay sweet potatoes at temperatures of 1° to 7° C. above freezing, and that at a higher temperature little or no decay takes place. It is not important as a storage organism, because sweet potatoes are stored at temperatures above those at which it will cause decay.

ALTERNARIA

Alternaria sp. has been isolated a number of times from sweet potatoes naturally infected when held at low temperatures. Inoculation experiments (*89*) showed that it produces a slow rot at about 7° C. or lower, but at a temperature of 20° only a very slight rot resulted after 19 days. However, in view of the temperature at which sweet potatoes are stored, there is very little danger that this fungus will become of much economic importance.

Alternaria sp. produces a firm moist rot. The tissue at first turns brown and then gradually darkens, but it never becomes black.

(Pl. 22, D.) The strands, so characteristic of soft rot when the potato is broken open, are not found in potatoes rotted by *Alternaria* sp.

PENICILLIUM

Penicillium sp. is a common inhabitant of decayed sweet potatoes. Like some of the other fungi already mentioned, it was most often obtained from potatoes held at low temperatures. While the results of inoculation experiments (*89*) show some slight success at low temperatures, this organism, even when removed from the competition of other fungi, must for the most part be considered a saprophyte.

Penicillium sp. forms blue masses of spores on the interior and on the surface of the sweet potato. (Pl. 23, B.)

BOTRYTIS CINEREA

Botrytis cinerea Pers. has frequently been isolated from sweet potatoes and other vegetables held at low temperatures. It is another of the organisms seldom encountered in sweet potatoes in storage, either because the conditions of the storage house are unfavorable to it or because it is unable to compete with other fungi. Inoculation experiments (*89*) have shown that it will decay potatoes slowly. When inoculated potatoes were exposed to different temperatures a more rapid and complete decay was obtained at temperatures of 7° to 14° C. than at higher or lower ones. However, it seems to cause decay over a much wider range of temperatures than some of the other storage-rot organisms.

B. cinerea produces a grayish, soft, somewhat watery rot (pl. 22, C) with a starchy odor. The affected tissue is more or less stringy when pulled apart. A slow decay is produced, requiring from 16 to 30 days to rot potatoes completely, depending upon the temperature at which they are held.

EPICOCCUM

Epicoccum sp. is not of much economic importance, although it has been isolated often from rotted sweet potatoes held at low temperatures. It grows rather slowly and is probably able to cause decay only when the competition of other fungi is removed. As a result of inoculation experiments (*89*), in which the potatoes were exposed to different temperatures, it was found that a little decay took place at 7° C. At other temperatures the potatoes remained sound.

Epicoccum sp. (pl. 22, A) produces a slow, firm rot. The tissue is slightly yellowish at first and later changes to a reddish brown color.

GIBBERELLA SAUBINETII

A number of species of Fusarium have been isolated from sweet potatoes in storage but, strange as it may seem, those species most frequently isolated have never been shown to be parasitic. On the other hand, those species demonstrated to be capable of causing decay (*Fusarium culmorum*, *F. acuminatum*) are seldom found under natural conditions.

Like some of the other fungi previously discussed, *Gibberella saubinetii* (Mont.) Sacc. (figs. 21 and 22) was isolated from sweet potatoes held at low temperatures. Inoculation experiments (*89*) have shown that this organism will cause decay if the sweet potatoes are kept from four to six weeks at temperatures of 2° to 4.5° C. *G. saubinetii* was recovered in pure cultures from 67 per cent of the sweet potatoes inoculated in the above experiments. The controls were all decayed, but none with *G. saubinetii*. As the moisture escapes, the tissue becomes firmer and finally hard and mummified. The color of the decayed tissue is at first a chocolate brown, which later turns to a pinkish brown.

FUSARIUM CULMORUM

Fusarium culmorum Wr. (fig. 23) seldom occurs in storage. Experiments (*89*) in which inoculated potatoes were held for a considerable length of time at different temperatures demonstrated that low ones were conducive to decay. For example, all the potatoes were decayed at 10.6° C., but only 10 per cent at temperatures of 13.5° and 16.9°. Although the controls rotted, they were not decayed by *F. culmorum*.

FIG. 21.—Conidia of *Gibberella saubinetti*, showing range of size and number of septations, also an ascus. Conidia × 1,200; ascus × 600

Fusarium culmorum produces a distinctly characteristic rot and when once seen would never be confused with any of the other rots except that caused by *F. acuminatum*, from which it does not differ macroscopically. From three to six weeks are required for it to decay the potatoes completely. The tissue is rendered spongy but not watery, and in the early stages a faint reddish brown color, which turns later to a carmine red or maroon, is produced. As the potato dries out, some of the color is lost, and in the mummified stage it becomes a beautiful pink.

FUSARIUM ACUMINATUM

Fusarium acuminatum Ell. and Ev. emend. Wr. (fig. 24) has been shown by inoculation experiments (*89*) to produce a slow decay of sweet potatoes when held at low temperatures, very similar to that caused by *F. culmorum*.

TRICHODERMA KÖNINGI

Cook and Taubenhaus (*31*) found *Trichoderma köningi* Oud. associated with ring rot and soft rot. These investigators success-

fully infected sweet potatoes by inoculations with a pure culture of *T. köningi*. The writers have seldom isolated this fungus from wounds or decayed tissue where it seemed to be the primary cause of the decay. A somewhat similar rot was also produced by *T. lignorum* (Tode.) Harz.

The symptoms caused by *Trichoderma köningi* are described by Taubenhaus and Manns (*185*) as follows:

> In the earliest stages, the spots are circular and of a light brown color, with a tendency to wrinkle. The flesh is hard and water-soaked, brown in color, with a black zone in the region between the healthy and diseased tissue. The spot enlarges in all directions and eventually destroys the entire root.

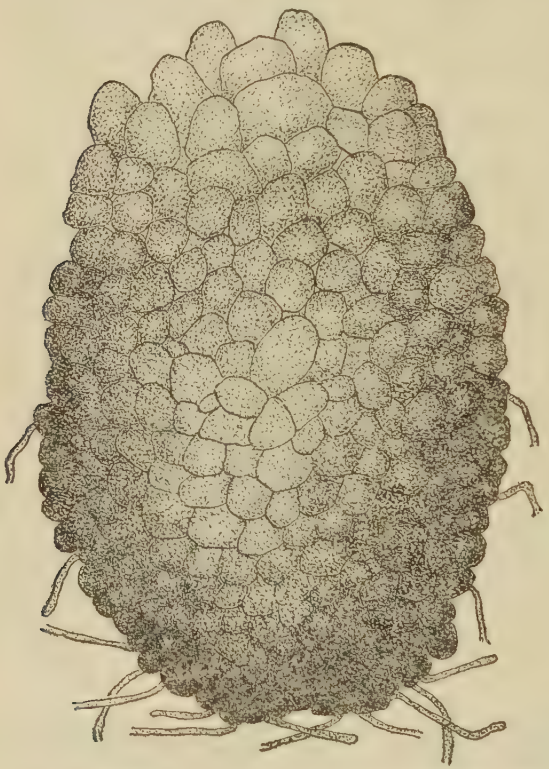


FIG. 22.—Mature perithecium of *Gibberella saubinetii* produced in a 28-day-old culture. × 500

SCLEROTINIA

Sclerotinia sp. seldom occur under normal storage conditions. If, however, sweet potatoes are held for several weeks at temperatures from 0° to 3° C. with a relatively high humidity, some of them may be more or less completely decayed by a species of Sclerotinia. The potatoes become soft and watery and large sclerotial bodies may develop over the surface. Sclerotinia rot has been observed on sweet potatoes in damp, cold cellars and in cold-storage houses where potatoes and other products requiring low temperatures are stored.

OTHER FUNGI

A considerable number of other fungi have been isolated from sweet potatoes in storage and studied, as, for example, *Fusarium batatatis* Wr., *F. hyperoxysporum* Wr., *F. radicicola* Wr., *F. solani* (Mart.) Sacc., *F. orthoceras* Appel and Wr., *F. orthoceras* var. *triseptatum* Wr., *F. oxysporum* Schlecht., *Nectria ipomoeae* Hals., and an undetermined species of Mucor.

Inoculation experiments have been made with all these organisms, and none of them have proved to be parasitic, although in some cases rather severe methods were employed. Special attention was given

to *F. batatatis* and *F. hyperoxysporum*, the two fungi causing stem rot. Both of these organisms invade the fibrovascular bundles of the potatoes. It is therefore of considerable economic importance to know whether or not these species cause storage rots, in view of their prevalence and destructiveness to certain varieties in the field. Stem-rot infected roots have been collected from the field and placed in storage, with the result that they kept just as well as healthy ones. In no case have the writers isolated one of the stem-rot organisms from sweet potatoes decayed in storage. From their results they feel justified in concluding that neither of these species causes a storage rot. Another species of Fusarium, *F. oxysporum*, which might be mistaken for *F. batatatis*, however, is frequently isolated from the stored sweet potatoes. In many houses potatoes may be found decayed for one-half to 1½ inches at the end, the tissue being brown and firm in texture and emitting a pleasant aromatic odor. (Pl. 22, E.) From such decayed ends and certain surface wounds and lesions *F. oxysporum* is generally isolated. Although the writers believe this species to be the cause of the end rot, they have failed consistently to obtain proof of it by any of the methods employed.

FIG. 23.—Conidia of *Fusarium culmorum*, showing range of size and number of septations. × 1,200

FIG. 24.—Conidia of *Fusarium acuminatum*, showing range of size and number of septations. × 1,200

Nectria ipomoeae, thought by Halsted to be the cause of ring rot of sweet potato and stem rot of the eggplant, has been isolated many times from decayed potatoes. Inoculations with this organism have failed to give any proof of its connection with any of the sweet-potato diseases.

A large number of other fungi have been isolated, with which no experiments have been made. Among others the following may be mentioned: *Zygorrynchus* sp., *Penicillium* sp., *Melanospora* sp., *Trichosporium* sp., *Ceratostoma* sp., *Sporotrichium* sp., *Pestalozzia* sp., *Aspergillus niger* Van Tiegh., *Sclerotium rolfsii*, *Fusarium vasinfectum* Atk., *Cephalothecium* sp., *Neocosmospora vasinfecta* (Atk.) E. F. S., *Verticillium cinnabarinum*, *Acromoniella* sp., *Macrosporium* sp., *Actinomyces* sp.

TRANSPORTATION AND MARKETING DISEASES

The principles underlying successful shipping and marketing are about the same as those for storage. It is, of course, more difficult to control conditions during transportation and on the market than in a well-constructed storage house.

The time required to transport sweet potatoes from the field to the market depends naturally upon their location and distance from each other. In summer sweet potatoes are frequently sent from southern Alabama, for example, to some of the northern markets, and many shipments are frequently made from Virginia to Kansas City, Denver, and other distant cities. Shipments over such distances often require two weeks or more. Since the organization of the inspection service of the Bureau of Agricultural Economics of the United States Department of Agriculture, reliable information as to the actual loss taking place during the shipment to distant points has been available. Inspection certificates issued by the bureau show that the losses vary from 9 to as much as 40 or 50 per cent, a 25 per cent loss being rather common. These certificates indicate that the principal losses are due to soft rot, black rot, and less commonly to dry rot.

Sweet potatoes are usually shipped from storage in the winter, when the weather is cold and subject to considerable variation, which means that the humidity and temperature in the cars are apt to be changeable and difficult to regulate. Before sweet potatoes are shipped they must be handled at the storage house and prepared for the market, the degree of preparation differing considerably in different localities. Some farmers grade their potatoes at harvest time and handle them as little as possible when preparing them for marketing. Others do most of the grading in the storage house during the winter just before shipment. In preparing the potatoes for shipment some growers clean and brush them by hand or by means of a machine constructed for the purpose, so that during the entire operation of grading, cleaning, crating, and hauling, some wounding is unavoidable. In view of the fact that wounding is necessary for infection by the soft-rot organism, it is not surprising that heavy losses are sustained from it every year. Since *Rhizopus nigricans*, the organism usually associated with soft rot, can completely rot a potato in from three to five days, a large percentage of loss may occur during the time required to ship the potatoes from storage to market. Besides soft rot and black rot, Java black rot may cause considerable damage, especially if the potatoes have been in transit for some time. Both these organisms develop rather slowly. Higher temperatures—that is, temperatures around 80° to 85° F.—are favorable for the growth of the black-rot fungus, and such temperatures may and probably do occur in the cars during transportation. Java black rot is more prevalent in shipments from the South. It is a slow-growing organism, but transportation from the Southern States to the northern markets often requires two weeks or more, thus giving ample time for considerable decay to take place.

A few other types of decay occasionally are found, such as dry rot and end rot, all of which cause appreciable loss every season.

The losses that occur after the potatoes reach the market and before they are consumed are caused by the same fungi as are found

during transportation, *Rhizopus nigricans* being the most destructive of the group. Each new handling produces fresh wounds through which the organisms enter, the result being that a large percentage of sweet potatoes decay between the time they are obtained by the merchant and the time they reach the kitchen.

These losses can not be entirely avoided. They can, however, be considerably reduced by the exercise of care in handling at each operation. It is advisable also during transportation and even on the market to keep the potatoes dry or as nearly dry as possible and to maintain a temperature of 50° to 55° F.

The facts to be emphasized are (1) that the potatoes should be carefully handled; (2) that a uniform temperature of 50° to 55° F. should be maintained; and (3) that the potatoes should be kept moderately dry.

In connection with the shipment of sweet potatoes over long distances, the results obtained by Taylor (*186*) are of interest. In a series of experiments in which the potatoes unwrapped and wrapped in newspaper and parchment paper were shipped to London from Virginia and New Jersey, it was found that they arrived in good condition. He came to the conclusion that freshly dug sweet potatoes could be delivered on the London market in a sound condition. He further proved that the unwrapped potatoes carried better than the wrapped ones, the latter showing a little waste.

DISEASE CAUSED BY NEMATODES

FIELD DISEASE

ROOT KNOT

Very little attention has been given to the occurrence of nematodes on sweet potatoes, although Bessey (*15*), who found that they penetrated some distance beneath the surface of the roots, lists the sweet potato as one of the crops on which they occur in abundance. In general, sweet potatoes are not severely injured by nematodes, though Elliott (*49*) cites a case where considerable damage was done in two counties in southern Arkansas, and O. C. Boyd found them on volunteer plants in fields of eggplant and tomato in Georgia. Root knot is quite common on sweet potatoes in the South and in parts of California, especially in the light sandy soils. The greatest loss, however, is not to the sweet potato itself, but to susceptible crops, like cotton or watermelons, which follow sweet potatoes in crop rotation. The nematodes seem to find the sweet potato a congenial host in which to live and multiply always without causing any great harm to the crop.

The root-knot disease is caused by a parasitic eelworm, *Caconema radicicola* (Greef) Cobb. It attacks a great many different hosts, making its control extremely difficult. Two methods, both more or less effective, should be employed in its control—(1), rotation with immune crops, such as some of the cereals, peanuts, etc.; and (2), planting of or, if necessary, the development of resistant varieties or strains.

Weimer and Harter (*202*) found nematodes quite prevalent on the roots of some varieties of sweet potatoes in the hotbeds in southern

California. Conspicuous galls (pl. 24, C) developed on the roots of such varieties as Nancy Hall and Red Brazil. The root knot was observed throughout the summer to cause dwarfing of the plants and consequently a reduced yield on the light sandy soils. A field test of the comparative susceptibility of a few varieties was made (*202*) at three locations in southern California in 1924. The results showed that the Nancy Hall and Red Brazil were very susceptible to root-knot injury, and large conspicuous galls developed on the roots. Such varieties as the Red Jersey, Yellow Jersey, Big-Stem Jersey, Porto Rico, Southern Queen, and Yellow Yam, while not entirely immune, are highly resistant and may be used in place of some of the susceptible varieties of sweet potatoes or other susceptible crops on nematode-infested soils.

PHYSIOLOGICAL DISEASES AND DISEASES OF UNKNOWN CAUSE

FIELD DISEASES

MOSAIC

In 1919, in Arkansas, Ensign (*54*) observed an abnormal condition of the Nancy Hall variety, which he identified as mosaic. (Pl. 16, B.) He described the plants as being more or less dwarfed, malformed, the leaves mottled. The difference in yield of healthy and diseased plants amounted to about 300 per cent.

Subsequent to Ensign's brief account of sweet-potato mosaic the disease was reported from Florida (*197*), Tennessee (*159*), and through the Plant Disease Reporter for 1923 from Texas and Kansas.[7] In 1923 the writers studied sweet-potato diseases in the San Fernando Valley of California, where mosaic occurred on a small percentage of the plants of the Nancy Hall variety. It was also seen by the writers in southern Mississippi in 1925. So far as is known the disease has not been observed on any other variety. From these few reports from widely separated regions it should occasion no surprise to find it wherever the Nancy Hall is grown.

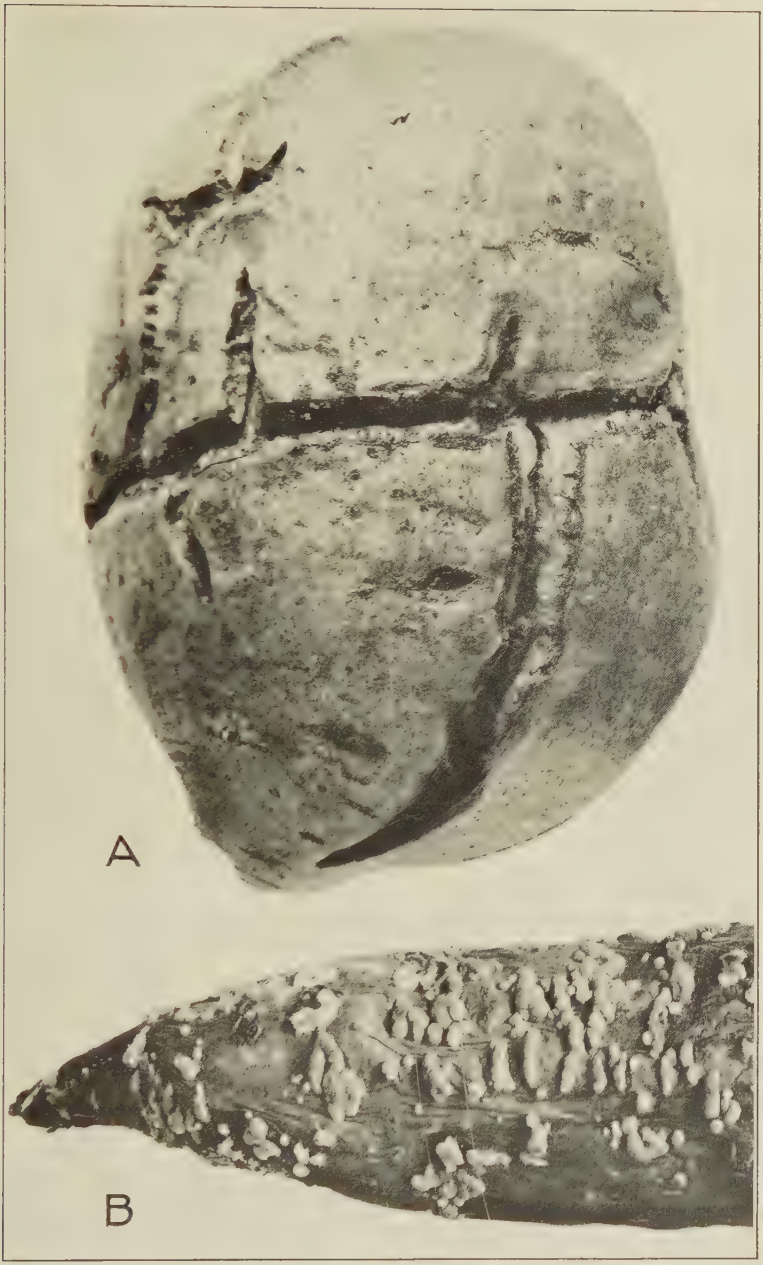
The total loss caused by mosaic is certainly small. Ensign (*54*) and others (*159*, *197*, *147*) report a considerable reduction in yield of affected plants. Weber (*197*) reports 6 per cent of the plants diseased in a 10-acre field and a yield of less than 10 per cent of normal. In California 1 to 3 per cent of the plants in a 20-acre field were diseased.

SYMPTOMS

Weber (*197*) has effectively described the characteristic symptoms of the disease as follows:

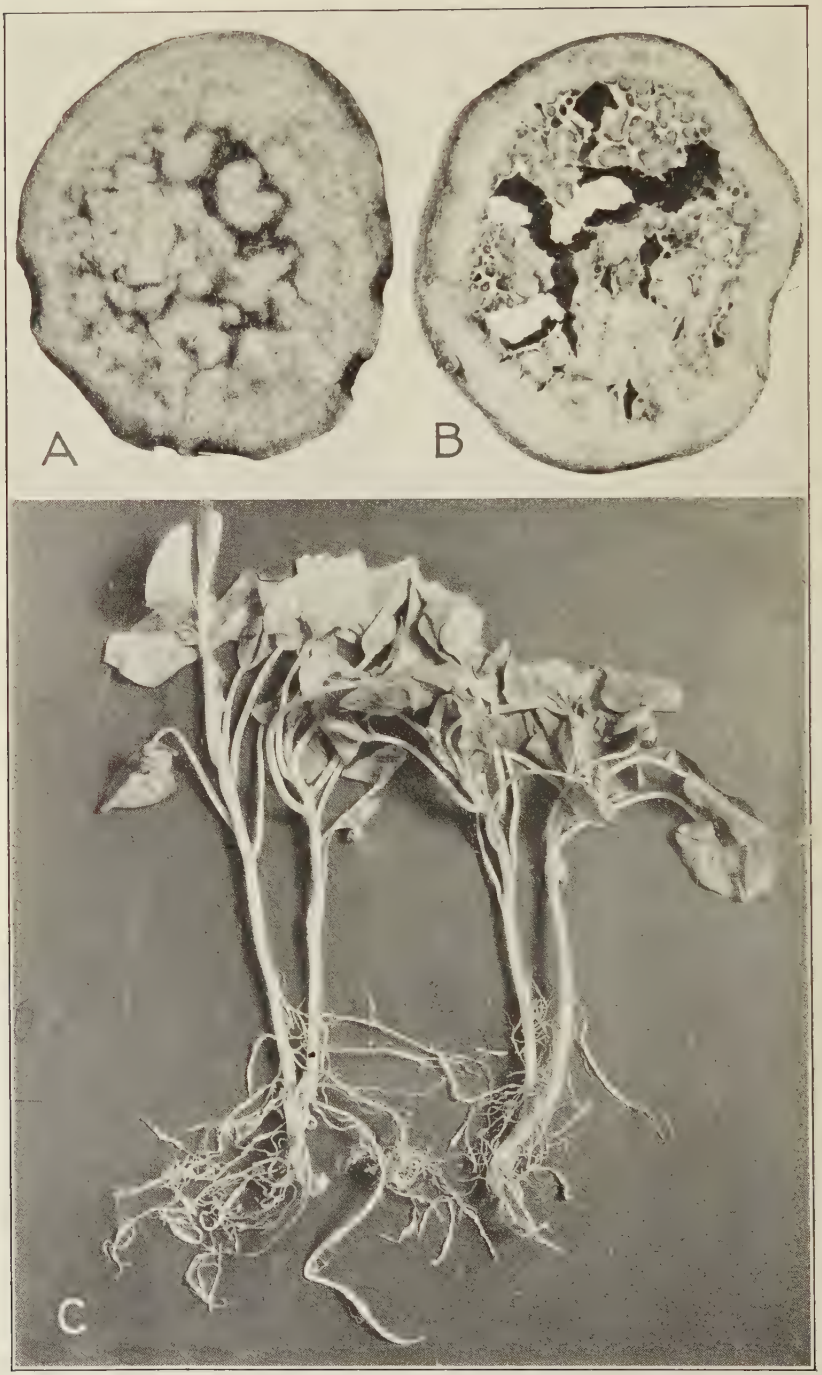
> Internodes decidedly shortened; thus the nodes bearing leaves are close together, affected plant showing a decided rosette appearance. The petioles are somewhat shorter and thicker than those of normal plants. The petiole at point of union with the blade is often flattened. The larger veins branch out into the blade abnormally suggesting fasciation and appear much wider and more

[7] UNITED STATES DEPARTMENT OF AGRICULTURE, BUREAU OF PLANT INDUSTRY. SWEET POTATO MOSAIC APPEARS IN KANSAS. U. S. Dept. Agr., Bur. Plant Indus. Plant Disease Rptr. 7: 71. 1923. [Mimeographed.]



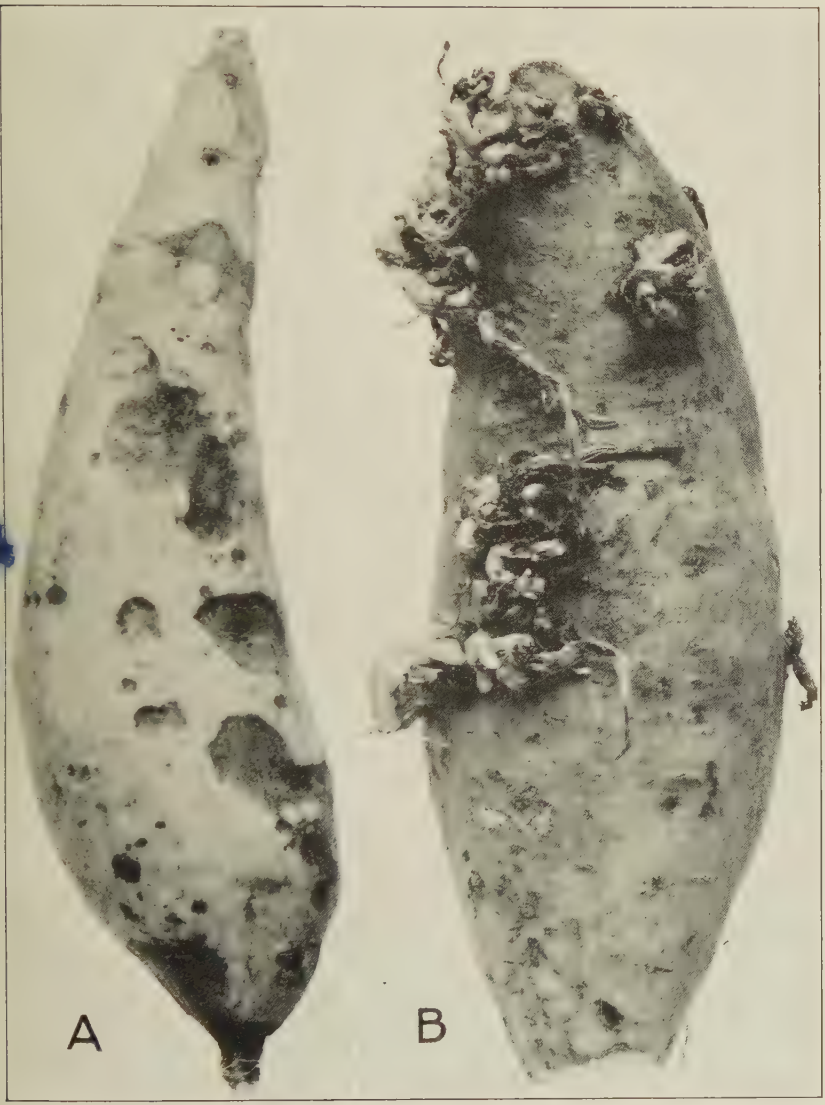
TWO DISEASES OCCASIONALLY ENCOUNTERED

A.—Growth cracking. Its cause is not known. It has been attributed to several things, such as wet weather following dry, and to too much nitrogenous fertilizer. B.—*Penicillium* sp. Note the cushions of the fungus over the surface of the potato. Penicillium appears usually only when the potatoes are held at a low temperature



STORAGE AND FIELD DISEASES

A.—Early stage of internal breakdown. B.—Advanced stage of internal breakdown. C.—Root knot caused by nematodes (*Caconema radicicola*)



MECHANICAL INJURIES TO ROOTS.—I

A.—Sunken spots caused by immersion in a 1 to 200 solution of formaldehyde for 30 minutes. B.—Sprouts killed in the hotbed by excessive heat from the sun and from manure

MECHANICAL INJURIES TO ROOTS.—II

A.—Irregular sunken spots caused by the application of fertilizer while the potatoes were still in the field. B.—Sunken spots caused by immersing potatoes in a 1 to 500 solution of mercuric chloride for two hours

prominent than the veins in normal leaves. The tissue between the veins is of a uniform green color, decidedly raised or sunken, forming pouch-like areas. There is no mottling, in the sense that leaves of cucumber and tobacco plants are mottled by mosaic. The blades are crinkled along the edges, curled backward and in severe cases malformed.

Cause of Mosaic

The cause of sweet-potato mosaic still remains somewhat of a mystery. In fact, the disorder is so different from the mosaic of many other crops that it seems questionable whether it should be so classed. Roots from plants exhibiting typical symptoms of the disease will yield mosaic plants, but not until recently has anyone succeeded in communicating it to other plants by hypodermic injections, by mechanical rubbing, or by grafting. Weber (*197*), Weimer (*199*), and Rosen (*147*) all report unsuccessful attempts to communicate the disease from mosaic to healthy plants. Weber attempted unsuccessfully to inoculate a number of plants by the injection of the juice from diseased ones, and Weimer, employing several different methods, such as grafting, hypodermic injections, and mechanical injury, failed to transmit the disease. More recently, however, Rosen (*148*) has obtained mosaic symptoms in inoculated plants, both by rubbing leaves of healthy plants with macerated mosaic foliage and by the injection of juice of diseased plants into the stems of healthy ones. No symptoms were obtained by him during the period covered by a single growing season or by three successive growing seasons in which the fleshy roots were used for propagation after the lapse of the intervening winter seasons. On the other hand, Rosen found that, if the vines were kept growing continuously 12 to 13 months or more, typical symptoms of the disease would appear in some of the plants. The plants were kept growing continuously vegetatively by making vine cuttings from inoculated plants, growing them in the greenhouse during the winter months, and setting them in the field the following spring. Thus, if inoculations were made in June of one season, typical symptoms in these plants should appear a year from the following July or August, or later. According to Rosen, the symptoms, mild at first, gradually increase in intensity until a certain maximum is reached. He also found that not all runners of the same plants would become diseased.

So far no evidence has been submitted to show that when vines are inoculated with the virus, if such it be, it is transmitted to the roots, which in turn produce typical mosaic vines. In other words, the roots have not been shown to play any part in the transmission of the disease from inoculated plants, although it is well known that roots from what may be called naturally infected mosaic plants do transmit the disease to the plants produced by them. There are, then, some differences hard to reconcile between inoculated and naturally infected plants. Out of the hundreds of naturally infected mosaic plants observed and studied by the writers no case has been found where only a part of the runners were diseased. They have further found that a mosaic potato will give only mosaic plants. It is not unlikely that further investigations may clear up some of these mysteries. It is barely possible that the roots of inoculated plants kept growing vegetatively for several years may give mosaic plants.

That the disease does not appear in all the runners is not necessarily surprising. Sweet potatoes frequently produce a number of long runners, and it would not be surprising if the virus failed to reach the ends of all of them during a single season when inoculated into the petiole of a leaf at the distal end of a vine. It is hoped that additional investigations will be conducted which will establish definitely whether or not the malady is a true mosaic, and, if so, the exact nature of it.

GROWTH CRACKING

The cracking of sweet potatoes resulting in one or more longitudinal or cross fissures or both (pl. 23, A.), one-fourth to one-half inch deep and as wide, has been found in many parts of the United States.

The cause of cracking, which is much more prevalent during some seasons than during others, is not definitely known, but evidence collected from growers and others indicates that it may in some way be associated with weather conditions.

It is believed by some investigators that cracking is likely to result when rapid growth is resumed after a temporary check brought about by drought or other causes. Others believe that cracking is a result of heavy feeding with a nitrogenous fertilizer, and still others insist that it is inherent in the plant. The latter theory hardly seems applicable to the sweet potato, however, since no instances are known where splitting has occurred every year, although the same seed stock has been used.

Some varieties are said to growth crack worse than others. G. P. Hoffman, former horticulturist of the South Carolina Agricultural Experiment Station, believes that the Georgia Buck variety is more subject to growth cracking than any of the others. Hoffman found that the application of stable manure was liable to stimulate growth cracking. J. A. McClintock observed that the Porto Rico variety was subject to splitting and that stable manure and nitrogenous fertilizers tended to produce it.

In 1922 the writers undertook a series of experiments that yielded some suggestive data on the comparative susceptibility of 18 different varieties grown near Rosslyn, Va. So far as precipitation was concerned the season was especially favorable for investigations of this type, the depth of rainfall, in inches, for June, July, August, September, and October being 4.14, 7.66, 4.09, 7.65, and 0, respectively.

The plants were set in the field May 31 and June 1. The soil was a rather heavy loam. The field sloped in such a way that one side was rather wet during a considerable part of the summer and especially for several days following heavy rains. The field was divided into three sections, and two rows of each variety were planted in each. The rows ran from the high to the low ground. No fertilizer or stable manure were applied. Sweet potatoes were grown on the ground the previous year.

There was an abundant growth of vines and a fair yield of potatoes. When the potatoes were dug October 2, 1922, careful notes were taken of the extent of growth cracking. Table 1 shows considerable variation among the different varieties with respect to growth cracking. Ten varieties were entirely free, while three, namely, Pumpkin Yam, Belmont, and Southern Queen, were conspicuously growth cracked. One significant fact observed in con-

nection with these investigations is that growth cracking was most prevalent on the better drained side of the field, which would seem to indicate that a very wet soil is not necessarily required to produce it.

TABLE 1.—*Comparative extent of growth cracking in different varieties of sweet potatoes*

Variety	Extent of growth cracking	Variety	Extent of growth cracking
Belmont	Considerable.	Nancy Hall	None.
Big-Stem Jersey	Slight.	Pierson	Slight.
Dahomey	Very little.	Porto Rico	Some.
Dooley	None.	Pumpkin Yam	Considerable.
Early Belmont	Some.	Red Brazil	Very little.
General Grant Vineless	None.	Southern Queen	Considerable.
Gold Skin	Do.	Triumph	None.
Haiti	Do.	Yellow Jersey	Do.
Key West Yam	Do.	Yellow Strasburg	Do.

The writers do not know at what time growth cracking started. There was no rain from September 15 up to the time the potatoes were dug, 17 days later. However, there was an abundance of moisture in the soil when the potatoes were harvested, so that the condition observed can not be attributed to drought. The same varieties were grown on the field in 1921, a season with a smaller amount of rainfall, but no growth cracking occurred. From these facts alone it would seem that soil moisture may be one of the factors associated with growth cracking.

FASCIATION

Fasciation in sweet potatoes is so general that it is sometimes difficult to find a plant in which it is not present in one form or another. Two distinct types are recognized, namely, flat fasciation (pl. 17, C) and ring fasciation, the former being the more prevalent. On the vines affected with flat fasciations they vary from slightly oval to broad bands 1 to 2 inches in width. The number of leaves per unit length on fasciated vines is often increased and frequently to such an extent at or near the growing point as to resemble a rosette. Occasionally fasciation begins soon after the sprouts are formed in the seed bed. It is more frequently observed, however, to originate at some little distance from the hill. The fasciated vine frequently increases in width with the increase in length.

According to Conard (*29*), ring fascination is much less common in sweet potatoes, occurring only in about one-half of 1 per cent in abnormal stems. This type of fasciation is round instead of flat, the vine being hollow. The leaves are numerous, and the phyllotaxy loses its regularity. The tubelike vines vary in size from one-fourth to one-half inch or more in diameter. Conard made a detailed study of the histology of flat and ring fasciation and discovered some very curious phenomena with respect to their formation. Those interested in this phase of the investigations are referred to his article (*29*).

No one seems to have satisfactorily explained the cause of fasciation. Different investigators have offered different theories, none of

which can be entirely accepted. Fasciation is more prevalent where there is a rank vegetative growth and where the soil is rich in nitrogenous fertilizers. Conard concludes from the evidence at hand that fasciation in the sweet potato is associated with high nutrition. De Vries (*196*) claims that it occurs mostly in plants subjected to conditions of nutrition above normal. Meehan (*121*) maintains its possible connection with disease. There are other investigators (*33*, *124*) who claim a connection between fasciation and mechanical injury. With respect to the cause of fasciation in the sweet potato, Conard writes as follows:

> An injury to the growing tip has been suggested as a cause of ring fasciation, but no sign of such has been found in any of the cases examined. We may therefore consider that whereas plain fasciation occurs when the meristem is so stimulated (by overfeeding or otherwise) as to cause it to spread out in two opposite directions and become linear at the apex, ring fasciation occurs when the same stimuli, operating for a time in radial symmetry, cause a spreading of the meristem in all directions equally, giving rise to a circular apical region.

INTUMESCENCE

Intumescence, or the formation of wartlike outgrowths or malformations on the upper and lower sides of the leaves, has been noted in a number of plants. A large assortment of explanations as to the causes of intumescence has been offered by different investigators; as, for example, by Sorauer (*164*), Atkinson (*9*), and Noack (*127*). Some hold that it is largely caused by excessively high temperatures, combined with a high relative humidity and impaired transpiration. Several investigators maintain that a reduced illumination stimulates the formation of intumescence, but that a high relative humidity contributes most to its formation. Dale (*34*) found that white light, or, in particular, the yellow or red rays, are absolutely essential for the production of intumescence.

Trotter (*191*), who studied the formation of intumescence of the sweet potato, found that it occurred only on the upper surface of the leaf. He noted the following histological variations: The cells of the epidermis change by gradual hypertrophy until they finally become of a height equal to or even greater than that of the thickness of the leaf. Such cells are irregularly distorted, their walls being thin and of a brown color and lacking or almost lacking protoplasm. He found that the intumescence of sweet potatoes occurred when the plants were subjected to an exceptionally high relative humidity and moderate light. He claims that excessive humidity is an indispensable factor in the formation of intumescence, but that such a definite statement could not be made with respect to light. High humidity seems to be the contributing cause, according to many investigators. However, Von Schrenk (*153*) has shown that such malformations may be produced by spraying cauliflower with certain copper fungicides. Wolf (*209*) found that mechanical injury, such as the beating of sand against the leaf, would cause it, and Smith (*162*) found that intumescence in cauliflower can be induced by exposing the leaf to vapor of ammonia. The writers have observed intumescence of the leaves of sweet potatoes growing in the greenhouse conspicuously formed independently of any known mechanical or chemical stimuli. After the plants were exposed several days to reduced light in an

atmosphere with a high relative humidity the outgrowths or intumescences formed in great abundance on the leaf. From the results of investigations and observations by different workers, it appears probable that intumescences may be caused by any one of several factors or conditions or by a combination of factors.

SUN SCALD

Injuries to the vines and foliage of sweet potatoes caused by heat and light are sometimes very difficult to separate, and for that reason both will be considered together under sun scald. Heat and light injuries occur during periods of clear dry weather and most often on very light, sandy soils. The most outstanding examples of sun scald have occurred in the San Joaquin Valley in California. In this valley the sweet-potato soils are very light and sandy, and water is supplied by irrigation. Except during or immediately following irrigation there is a layer of dust about 2 inches thick on the surface which contains very little moisture. The temperatures in midday often reach 110° F. or more over a considerable portion of the summer, frequently beginning in May about the time or soon after the plants are set out. The sky is usually clear. At this stage the plants have very little foliage and no vines. The stems as well as the leaves are exposed to the direct rays of the sun, so that the temperature at the surface of the soil is extremely high. If there is a period of clear hot weather, the stem at the soil line, if exposed to the direct rays of the sun, is scorched, becoming gray to brown and frequently constricted to about one-third of its normal diameter.

Injury of a somewhat different character occurs on older plants with vines 1 to 3 feet or more in length. In this stage the vines are protected by the foliage, which turns first yellow and then brown, beginning at the margin of the leaf at the tip or just back of it. The entire leaf finally dries up (pl. 17, A) and can be crumbled into a powdery mass. The leaves finally fall to the ground and in severe cases form a brown, leafy carpet over the surface of the soil, leaving the vines more or less exposed to the direct rays of the sun. The tips of the vines, being more tender and succulent, are frequently killed, so that new branches are developed from the axils of leaves farther back. The plants, if not killed when very young, recover with the coming of cooler weather, or if irrigated so that the water comes to the surface at the top of the ridge. How much of this injury is due to light alone is not known. Experiments have shown, however, that leaves of sweet potatoes exposed to strong light and a moderate temperature first turn yellow and later become brown, a condition which was observed in a field of Nancy Hall near Bakersfield, Calif.

Sun scald has been observed to cause a similar injury to the foliage, although to a less extent, in some sections of the eastern United States. The leaves at the center of the hill often turn yellow and later become brown during or immediately following a period of cloudless hot weather. It frequently results in considerable defoliation for a distance of 1 to 2 feet from the center of the hill. What is probably the same trouble has been reported in South Africa by Doidge (*37*) as sun scorch.

STORAGE DISEASE

INTERNAL BREAKDOWN

Internal breakdown of sweet potatoes, which occurs during some seasons and in some varieties more than others, is apparently more or less correlated with the conditions maintained in the storage house. The cells or groups of cells of sweet potatoes that have been in storage for several weeks at a relatively low humidity and high temperature are frequently torn apart. In extreme cases the separation or mutilation of the tissues results in the formation of cavities of varying sizes visible to the unaided eye. In cross section (pl. 24, A) the tissue is somewhat cottony in appearance, the first cavity being found at the center. In extreme cases (pl. 24, B) internal breakdown can be detected by the sponginess of the potato when squeezed in the hand.

Internal breakdown is quite prevalent, specimens of this disorder having been received from various parts of the United States. Internal breakdown increases with the increase of time in storage, the largest percentage of specimens being present in the latter part of the storage period.

An examination of the affected areas shows that the cells are partly destroyed and that their starch content is somewhat reduced (*83*). Artschwager (7), in an anatomical study of the sweet-potato root, made also some detailed anatomical observations of internal breakdown tissue. He says with respect to the conditions found:

> From the study of the structure of the normal tuber, it will be recalled that the groups of vascular tissue are separated by undulating bands or areas of parenchyma. When tubers in the initial stage of breakdown are examined, it becomes evident at once that the first pathological disturbance has taken place in the interstitial parenchyma. The cells of this tissue are large, irregular, and poor in starch; vascular elements and latex tubes are entirely wanting. When breakdown occurs, the cells of the affected region become at first more or less dehydrated; this is indicated by their infiltration with air, giving them a pure white appearance whereby they stand out strikingly from the surrounding tissue. This parenchyma gradually acquires a spongy texture as the obliteration of cells progresses, leading finally to the formation of small, polyhedral chambers which are lined by the remnants of the destroyed tissue.

No organism has ever been isolated from breakdown tissue. It is believed that the disturbance is in some way correlated with environmental conditions in storage. A few inconclusive experiments have been conducted which suggest a possible explanation. Two varieties of sweet potatoes (Yellow Jersey and Southern Queen) were exposed in two automatically controlled incubators for three and one-half months at temperatures of 25° and 29.5° C. and at a relative humidity of 65 to 70 per cent, and also under storage conditions, as controls. At the end of the experiment each potato was examined. It was found that more breakdown had taken place in the incubators than in the storage house with a temperature of approximately 12° to 15°. No measurable difference in the amount of breakdown in potatoes exposed to temperatures of 25° and 29.5° was noted. There was, however, more breakdown in the Southern Queen than in the Yellow Jersey. Externally the potatoes were perfect, though they were slightly spongy when pressed. The evidence obtained from numerous

observations in storage houses and from a limited number of experiments indicate that internal breakdown probably results from exposure to high temperatures, combined with a relatively low humidity.

DISEASE CONTROL

FIELD DISEASES

Certain of the numerous diseases of the sweet potato, such as stem rot, black rot, foot rot, soil rot, scurf, Texas root rot, and root knot, are severe enough to justify the application of control measures. These diseases occur principally on the underground parts of the plants, on the stems near the soil line, or within the roots and stems. None of the diseases of the leaves are sufficiently severe to justify the application of control measures. The diseases in connection with which remedial measures are necessary are those in which the causal organism inhabits the soil as well as the potatoes themselves. This makes the problem of control on the whole more difficult.

It is not surprising that the first experiments on the control of sweet-potato diseases were made with fertilizers. In 1880 Dudley (*58*) made what probably constitutes the first published report of the occurrence of any sweet-potato disease in the United States, where mention is made of a rot that is said to be as destructive to the sweet-potato crop as in former years. It is not possible to state definitely which of the diseases was referred to, but it is believed to be soil rot, since it was said to be connected in some way with the ground. Some experiments were conducted with potash, but the disease was not lessened thereby. Experiments with commercial fertilizers and other substances, such as lime, gypsum, and sulphur, were conducted for a number of successive years for the control of soil rot and other diseases of the sweet potato by the New Jersey station and by Chester (*27*) in Delaware. The earliest investigations on the control of sweet-potato diseases in New Jersey were later repeated by Halsted, who conducted some very comprehensive field tests with various commercial fertilizers and chemicals for the control of soil rot and black rot. He only slightly reduced the amount of soil rot by the application of flowers of sulphur. On the whole, then, the application of fertilizers and other chemicals to the soil has not thus far adequately controlled any of the sweet-potato diseases. Further consideration of some phases of this subject will be given later.

Following Halsted's, no investigations looking to the control of sweet-potato diseases were undertaken until about 1911, when Harter, of the United States Department of Agriculture, and Taubenhaus and Manns, of Delaware, began a study of the several diseases. These investigators coupled their studies of the causal organisms with intensive field tests on control measures. The results of their investigations and later those of Poole (*133*) brought out the fact that chemicals could not be wholly relied upon to control the diseases, but that sanitary measures coupled with careful seed selection must be employed (*70*, *75*, *185*). Since the publication of the three papers just cited, a considerable number of additional experiments on the control of sweet-potato diseases have been conducted, the nature and results of which will be given in some detail.

For convenience in presentation, the control of sweet-potato diseases will be considered under the following topics: Exclusion from foreign countries; inspection and certification; eradication; and resistant and susceptible varieties.

EXCLUSION FROM FOREIGN COUNTRIES

It would be possible to cite several diseases that are of minor importance in foreign countries and that if introduced into the United States might spread rapidly and cause enormous losses to the crop. The fact that a disease may be of little or no economic importance in one country, but very serious in another, is, in part at least, a justification for the rigid enforcement of a quarantine against its introduction. To meet such possible emergencies a plant quarantine against the introduction of plants from certain countries is now maintained by the United States Department of Agriculture. Furthermore, all introductions from whatever country are carefully inspected before they are sent out for propagation. The introduction of both injurious insects and fungous diseases may thereby be avoided.

Probably none of the diseases of the sweet potato in the United States have been introduced. Up to the present time there has been very little introduction or importation of sweet potatoes into the United States, although there is some exchange of the plants or potatoes between this country and some of the islands of the West Indies. Some of the diseases are common to both the West Indies and the United States, but some of the more serious ones, stem rot and foot rot, for example, are not known to occur in the West Indies.

Sweet potatoes are grown widely throughout the tropical and semitropical world. The diseases in these countries are only imperfectly known, except in Japan, where the sweet-potato industry has been greatly developed in the last few years and now equals about three-fourths that of the United States. Considerable literature is published in the Japanese language, including at least one book dealing with sweet potatoes and their diseases, several of which have never been reported as occurring there. On the other hand, some diseases are common to both Japan and the United States, as, for example, scurf, caused by *Monilochaetes infuscans;* black rot, caused by *Ceratostomella fimbriata;* soil rot, attributed by the Japanese investigators to *Acrocystis batatae;* and probably stem rot, although from the literature it is not possible to determine this with certainty. One or two of the diseases reported only from Japan are said to be very destructive there.

In view of these facts it is highly important that the diseases of foreign countries which do not occur in the United States be excluded. In order to do this successfully it is necessary to know what the diseases are and on which hosts besides sweet potatoes they occur, which might be introduced by importations into this country.

INSPECTION AND CERTIFICATION

Although the diseases of the sweet potato are fairly widely distributed in the United States, there are localities in which they do not occur or are found only to a very limited extent. In recent years a considerable industry in the growing of plants for sale and shipment

has developed in the United States, which has resulted in the wider and more general distribution of some of the diseases. Potatoes are also sent long distances for seed purposes. The appearance of some of the worst diseases in some localities has been definitely traced to the shipment of plants or potatoes from a locality where the diseases were present.

To prevent the spread and introduction of these diseases some of the State legislatures have passed regulations requiring an inspection and certification of the seed stock and plants before they can be sold. In 1927 eight States had in operation quarantine regulations that prevented the importation and sale of plants that did not carry an inspection certificate. The authority for administering the inspections may be placed with the several State departments of agriculture or with the State agricultural experiment stations. The inspections are made for both insect pests and plant diseases at designated times by an entomologist, plant pathologist, or other duly authorized agency.

Unfortunately, the methods employed by the different States are not uniform. All or practically all States require one field, one bin, and one bed inspection, and some States more than one. There is likewise no uniformity in the percentage of diseased plants or potatoes considered permissible in the different States. Each State passes regulations that seem to meet its own ideas of local needs. Shipments from one State to another are therefore sometimes refused because the laws governing inspection and certification in the State of origin do not meet the requirements of the State to which the plants or seed potatoes are to be sent.

In spite of the lack of uniformity among the States with respect to seed certification and inspection, the disease situation has materially improved since the State regulations went into effect. Many of the plant growers have voluntarily set about cleaning up their crop, in order to gain the advantage of advertising certified seed potatoes and plants for sale. Others have been forced to do so for fear of being refused the right to sell their plants or potatoes for seed purposes. It is to be hoped that all States will pass regulations governing seed certification and that they will eventually become uniform throughout the United States.

ERADICATION

Seed Selection

Careful selection of disease-free seed potatoes, coupled with disinfection, clean seed beds, and crop rotations (*75*), offers the best possibilities for the control of sweet-potato diseases. Some of the fungi causing these diseases invade the roots and live over the winter in or on the potatoes in storage. They then grow from the diseased seed potatoes into or on the plants developed from them. In the early stages diseased plants are difficult, if not impossible, to detect; as a result, many are set in the field, where the fungi later develop. It is therefore imperative that only healthy potatoes be used for seed.

Seed potatoes should be selected in the fall at digging time while they are still attached to the vines, so that any that are diseased may be discarded. The stem just above the roots should be split

open and the seed potatoes taken only from plants whose fibrovascular bundles are not blackened by stem rot. Farmers sometimes insist upon making their selection in the winter or spring from the stored potatoes by cutting off the ends and rejecting any with discolored bundles. This is not a safe practice, however, for two reasons. (1) It is difficult and frequently impossible to tell whether the potatoes are diseased or not, since the fibrovascular bundles are often blackened after a few weeks in storage. The rejection of all potatoes showing any darkening of the fibrovascular ring would entail considerable more labor and handling than is actually necessary to obtain disease-free seed. (2) It is impossible to detect infections not sufficiently advanced to darken the fibrovascular ring, a condition that would apply in a lesser degree to selection in the field. Under field conditions the stem is likely to show infection when the potatoes would not, since the former is usually first invaded. The presence of the fungus would therefore be revealed by the blackening of the fibrovascular bundles of the stem before it would show in the roots. The selections of seed potatoes should be made in the field, if possible, before a heavy frost or very soon thereafter. The discoloring of the vascular ring of the stem and roots that occurs a day or two after a frost is dark, very much resembling the blackening caused by stem rot. This discoloration does not take place as promptly in the roots as in the vines. Therefore, if it is impossible to dig and select seed at once, the vines should be promptly cut. Doing this will prevent to some extent the discoloration of the fibrovascular bundles of the roots. The potatoes selected for seed should be stored in baskets or boxes and kept apart from the general stock.

Seed selection should be made for stem rot, black rot, scurf, soil rot, and foot rot. In the spring, just before bedding, the selected seed should be carefully picked over and any potatoes having black rot, scurf, foot rot, or, in fact, any of the field diseases should be discarded. It is especially important that this be done, since there are some diseases like black rot and foot rot that develop after the potatoes are placed in storage.

Seed Treatment

Seed treatment for the control of stem rot, black rot, scurf, and foot rot is employed for the purpose of destroying the spores of any of the parasitic fungi adhering to the surface. The hyphae or spores buried can not be killed by the treatment. Although treatment of seed sweet potatoes in a solution of mercuric chloride (bichloride of mercury, or corrosive sublimate, as it is frequently called) has been recommended for the control of scurf, it gives only a partial control at most. The hyphae of the scurf organism are buried in the skin, and consequently they are not killed by the mercury compound. The writers conducted experiments in which potatoes badly affected with scurf were treated for different lengths of time (10 and 30 minutes). After treatment the potatoes were bedded in sterile soil, and the slips produced from them were set in soil on which sweet potatoes had never been grown. The results showed that only partial control resulted from a 30-minute treatment.

Additional experiments also showed that the scurf organism on the slips could not be destroyed by immersing the young plants for 5 and 10 minutes in some of the common fungicides, as, for example, mercuric chloride (1 to 1,000), formaldehyde (1 to 240), lime-sulphur (1 to 50), and Bordeaux mixture (5–5–50). The roots, as well as some of the leaves, were badly injured by some of these chemicals, and many of the plants died soon after they were set in the field. The greatest injury resulted to those plants dipped in the formaldehyde solution, a 5-minute immersion being so injurious as to be prohibitive. When the potatoes were harvested there was more or less scurf on the plants dipped in all the different solutions. These results show that scurf can not be adequately controlled by either treating the potatoes alone or by immersing the plants in certain fungicides. Any treatment long enough or severe enough to kill the organisms within the host would, at the same time, seriously injure the potatoes. The treatment of sweet potatoes for 5 to 10 minutes in a solution of corrosive sublimate (1 to 1,000) reduces the germinating power of the potatoes slightly, but at the same time more vigorous plants are usually produced. The potatoes should be bedded immediately after treatment; rinsing the potatoes in water or drying them before they are bedded is not necessary. Since mercuric chloride reacts upon iron or tin, and its disinfecting power is thereby reduced, only wooden or enamel vessels should be used. A bushel crate or basket serves as a convenient receptacle for use in dipping the seed. Grain sacks and gunny sacks, which remove a considerable portion of mercury from the solution, should not be employed.

For ordinary farm practice a 63-gallon barrel about half full of the disinfectant answers the purpose very well. If larger quantities of potatoes are to be treated, a wooden tank holding several hundred gallons or several barrels in which as many lots of sweet potatoes can be treated simultaneously may be used. A method has been worked out whereby the solution may be used a number of times for treatment by the addition of a small quantity of the corrosive-sublimate crystals and enough water to bring the solution up to its original volume. The crystals should be dissolved in hot water before being added to the solution, as they dissolve very slowly in cold water. Convenient mercuric-chloride tablets containing enough mercury when dissolved in a pint of water to make a 1 to 1,000 solution are now on the market. These tablets also contain ammonium chloride or citric acid (which assists in the ready dissolution of the mercury without the use of hot water) and a blue dye that colors the solution.

For the commercial treatment of sweet potatoes the following general recommendations should be used as a guide. These recommendations are based on the supposition that the potatoes are reasonably free from dirt, which has been shown to remove or fix some of the mercury. An average of approximately 5 per cent of the mercury is removed by each 5 bushels of potatoes reasonably free from soil and treated in hampers in a clean barrel containing 32 gallons of solution. In view of this fact, the following recommendations (*198*) may be made:

After each 10 bushels of potatoes treated, add two-fifths to one-half ounce of mercuric chloride and enough water to restore the solution to its original volume. The solution should be discarded after the treatment of about 50 bushels. If

a tank holding a larger volume of solution is employed, the proportions of mercuric chloride and water to be added should be calculated in accordance with the quantity of solution originally used and the quantity of potatoes treated at one time.

After a number of months in storage the spores of Rhizopus on the potatoes are generally very numerous. Even under the best possible storage conditions some of the sweet potatoes often are rotted by this fungus. It fruits readily and produces spores in great abundance. It is not unlikely therefore that many spores are present on every potato in the storage house. These spores germinate in a few hours (*201*) under favorable conditions of temperature and moisture, with the result that the potatoes are frequently rotted in the hotbeds without producing any plants. Investigations have shown that seed treatment with corrosive sublimate lessens the chances of decay in the seed bed.

SEED-TREATMENT INJURIES

The treatment of seed sweet potatoes by immersion in a solution of mercuric chloride, formaldehyde, or any of the phenol-mercury compounds tried for even five minutes is likely to be followed by some injury, which increases with the length of the treatment and the concentration of the solutions. Formaldehyde apparently causes more injury (pl. 25, A) than mercuric chloride (pl. 26, B), and for that reason is not recommended. The injury seems to be caused by the disinfectant penetrating the potato through wounds caused by bruising and through lenticels and dead rootlets. The cells thus killed collapse, leaving from a few to many small more or less circular pits 1 millimeter to 1 centimeter or more in diameter.

PREPARATION AND MANAGEMENT OF THE SEED BED

The use of clean seed beds will help to reduce the black rot, foot rot, scurf, Sclerotium rot, Rhizoctonia, and possibly to a limited extent the stem-rot diseases. The preparation of the bed should receive attention primarily from the standpoint of sanitation. Almost any method for its preparation that guarantees freedom from disease-producing organisms may be used. The repeated use of the same soil year after year in the seed bed is one of the chief means of perpetuating sweet-potato diseases. After the plants are all pulled from the bed the soil often is either left in the beds or thrown to one side with all the decayed potatoes and manure. The organisms contained therein multiply, and if the soil is used again the following year these serve as sources of infection for the new crop. If this soil is simply thrown to one side, many of the parasites contained therein find their way back into the new bed. It is therefore imperative that the soil or sand from the old beds be disposed of and replaced with new soil or sand for the new crop. Before new soil is added the framework should be disinfected by thorough spraying with a solution of formalin—1 pint to 30 gallons of water. This treatment should be repeated after 24 hours, if possible. If more convenient, a solution of copper sulphate—4 pounds to 50 gallons—may be used instead. It makes no difference in this operation what disinfectant is used. The rubbish and decayed sweet potatoes should be gathered up from around the bed and destroyed and the ground thoroughly sprinkled with a disinfectant.

The source of the soil or sand for the hotbed is of primary importance. The use of soil from fields where sweet potatoes have been grown should be avoided, if possible. A good quality of sand that does not form a hard crust will give better and stronger plants than soil and is much less likely to be infested with disease germs. When it is possible, the soil or sand should be obtained from the woods or from uncultivated fields. It is a good practice to throw off the surface 6 inches and use the subsoil. The same farm implements used to handle and haul away the old dirt should not be used to handle new soil or sand without first being cleaned and disinfected with a solution of either formalin or corrosive sublimate.

When other sources of heat are available for the seed bed the use of stable manure is not recommended, for reasons already mentioned. The chances of introducing the disease germs in the seed bed by employing manure to generate heat or to stimulate the growth of the plants by mixing it in the soil about the potatoes and plants is so great that the practice should be discouraged. Farmers sometimes hesitate to use pure sand in the seed beds, believing that no food is contained therein for the use of the plants. This question has been thoroughly considered by the writers, and experiments made by them have shown that better rooted plants can be obtained in sand than in rich dirt. The reasons for this are obvious. To understand why it is so, one needs only to remember that the mother potato can and does furnish all the food required by the young plants. In rich soil the development of a large root system would be unnecessary, since the plants as they grow older and begin to search for food and water outside of the mother potato are able to obtain all the plant food needed close at hand. In sand, however, food materials are scarce, and an abundant root system is developed in order to obtain plant food.

Objections to the removal of the soil from the seed bed every year are sometimes made, the farmers preferring to disinfect it instead. The writers strongly recommend the use of new soil, but if disinfection of the old soil is insisted upon formaldehyde should be used. The soil should be piled up and then soaked with a solution of formaldehyde, 1 pint in 20 gallons of water. The treated soil should be covered with a canvas or tarpaulin, to confine the fumes as much as possible and to prevent reinfestation with disease germs. Soil disinfected with formaldehyde can not be used immediately, because of the danger of injuring the potatoes. A week or more should elapse after treatment before the soil is used in the seed bed. Meanwhile frequent stirring should be given to assist the gas to escape. The soil should never be treated while it is spread over the bed, especially if stable manure is used under it to generate heat. With the idea of reducing the labor to a minimum, farmers have treated the soil in the bed and at the same time have soaked the manure below. This naturally sterilized the manure, rendering it useless as a source of heat, resulting finally in the entire loss of the bed from the rotting of the potatoes. In view of the cost, labor required, and risk to be taken, this method is not recommended unless carried out under expert supervision.

Notwithstanding the fact that sand is superior to soil in which to bed sweet potatoes, care must be taken to prevent the bed from getting

too hot and dry. Sand dries out rapidly and upon exposure to the direct rays of the sun may get hot enough to burn the young sprouts before they emerge from the soil. (Pl. 25, B.) This danger can be averted by shading the bed with canvas or by other means during the hottest part of the day or by keeping the sand moist at the surface by the application of a small quantity of water. The temperature about the potatoes in the hotbed should be maintained as nearly as possible at 75° to 85° F. A temperature of 90° is not dangerous, and sweet potatoes will endure 100° for a short time. After the potatoes have germinated, the temperature should be lowered and the cover removed from the bed in the daytime during fair, warm weather, in order to prevent the plants from growing too rapidly and becoming too succulent and tender. In other words, sweet-potato plants should be hardened before they are planted in the field.

The application of stable manure or of mineral fertilizers as a top-dressing to sweet-potato seed beds is not advisable. The mother potato contains all the nourishment required to grow plants large enough for planting, and if the sand or soil is poor in nutrient material better rooted plants are developed than when food materials in the form of stable manure or mineral fertilizers are close at hand. Sodium nitrate applied alone or in combination with other food elements if not judiciously used may cause injury to the young sprouts before they emerge from the soil and even to the potatoes themselves. (Pl. 26, A.) Injuries to the plants and potatoes caused by the injudicious application of fertilizers have often been attributed to some disease such as black rot. The growing tips of the young sprouts before they emerge from the soil are very succulent and tender and are easily injured or even killed by a dilute solution of sodium nitrate. The sprouts seldom completely recover from chemical injuries. The potatoes in the bed are less likely than the sprouts to be injured by the application of sodium nitrate or of a complete fertilizer. When the potatoes are injured the injury usually occurs on the upper surface in a somewhat irregularly shaped water-soaked region one-eighth to one-fourth of an inch in depth. Such organisms as *Rhizopus nigricans* may gain a foothold in such wounds and cause a complete decay of the potato.

Slip Seed

Slip seeding as practiced in the South, where there is a long growing season, can be recommended for the following reasons: (1) The quantity of potatoes required for bedding is greatly reduced; (2) it is an efficacious means of controlling some of the important diseases when intelligently applied; and (3) better yields are claimed and frequently are obtained.

Briefly the methods of slip seeding are about as follows: A quantity of potatoes, the number depending upon the acreage to be planted, are bedded early in the spring. When the plants in sufficient numbers are large enough to pull they are set out to provide slips for planting the main crop. If conditions are favorable, by May or June each plant has produced several long vines, which are cut into sections to include about two nodes and set in the field with the end nearest the hill below ground. Many acres may be set in

this way. The plant develops roots in a short time and by the middle of October or the first of November will produce a full crop.

Slip seeding is hardly possible in the northern sweet-potato belt because of the shortness of the season. When intelligently done it is an efficacious means of controlling and eradicating sweet-potato diseases. The requirement that healthy plants from the seed bed must be set in soil free from disease-producing organisms applies also to vine cuttings. Experiments have shown that a large percentage of the individual cuttings become diseased if they are planted on soil infested with the stem-rot fungi. The same would doubtless hold true for black rot, for the reason that the entrance of the parasite is greatly favored by the wound made in cutting the vines. The writers have examined quantities of slip-seed stock both in the field and in storage and found an abundance of black rot, stem rot, foot rot, scurf, and whatever diseases are known to the locality.

To obtain results from slip seeding, the following precautions must be taken: (1) The cuttings should be made from healthy vines. This will seem obvious when it is remembered that the stem-rot organisms often grow into the vines 8 feet or more from the hill and that it is necessary sometimes to break open the cortex in order to detect it. (2) The cuttings should be planted on new ground or on ground that has not grown sweet potatoes for at least six years. (3) The potatoes from cuttings must be picked over and disinfected in the spring before being bedded, according to directions already given. (4) The potatoes must be bedded in a bed prepared according to directions given above.

Fertilizers and Soil Treatment

Lime and gypsum have been used on soil infested with the stem-rot fungus without noticeable difference in the proportion of disease in the succeeding crop. Fertilizers are not believed to exercise any control over stem rot, although the writers have not personally given attention to this phase of the problem. It was thought to be of such doubtful possibility that fertilizers were disregarded in searching for control measures. That these predictions were not wholly unfounded can be inferred from the work of Jones and Gilman (*101*), who, as a result of extensive experiments with fertilizers, came to the conclusion that they were valueless in controlling the yellows of cabbage caused by *Fusarium conglutinans*, a disease very similar to the stem rot of sweet potatoes. Instead of decreasing the disease, mineral fertilizers have been shown to increase the severity of stem rot. Poole (*136*) found that a fertilizer consisting of ammonia 2 per cent, phosphoric acid 8 per cent, and potash 7 per cent, when applied broadcast in the row, if not dissolved by rains before planting, caused injury to the roots through which the stem-rot organisms entered. Kainit as a source of potash was found to cause more injury than the muriate. No injury resulted from the use of sulphate of potash.

Undissolved fertilizers in contact with the roots cause dwarfing of the plants. The vines are short or wanting, the plants growing more or less erect and rigid. The internodes are abnormally short (one-fourth to three-fourths of an inch), leaves bronzed, rather crisp and small (about one-third normal size), with short petioles. No fleshy roots are produced, the fibrous ones being apparently normal. The

plants are often killed by fertilizers, but those which survive often revive and yield a fair crop following a rain sufficiently heavy to dissolve the potash. In some of the earlier investigations Halsted and Chester, in New Jersey and Delaware, respectively, applied different fertilizers for the control of black rot and soil rot, without any outstanding beneficial results. Halstead found that the application of the flowers of sulphur decreased soil rot, the yield of potatoes free from soil rot being proportional to the quantity of sulphur applied. Grantham and Manns (*58*) showed, on the other hand, that lime increased soil rot. In general, it has been fairly well demonstrated that substances tending toward acidity decrease soil rot and alkaline ones increase it. The flowers of sulphur were tried by Selby (*154*) as a control for soft rot. He obtained no control, but it was found that smoother potatoes were obtained.

Soil rot is a disease especially difficult to control. Experiments conducted by the New Jersey Agricultural Experiment Station as early as 1880 (*38*) on the control of what the writers believe to be soil rot showed the application of fertilizers to be ineffective. Experiments by Halsted and by others in more recent years have shown that the use of fertilizers and chemicals applied to the soil gives very little control, although it was found by Halsted, Poole, Adams, and others (*114*) that the application of the flowers of sulphur somewhat increased the yield of clean roots, the increase being in proportion to the quantity of sulphur applied up to about 300 pounds to the acre.

Experiments conducted by the writers in New Jersey for two years showed that the application of chemicals gave no control. These experiments were repeated in Delaware in 1919 and 1920 on a 1-acre field that had been in sweet potatoes consecutively for 13 years and was so badly infested with soil rot that a crop could not be grown. Stable manure, lime, potash, phosphate, sulphur, table salt, and other substances were used alone or in combination without giving control. The yield was slightly better where stable manure was applied. This was due to the stimulation of growth rather than to a reduction in the percentage of soil rot.

In recent years considerable interest has been aroused in the use of inoculated sulphur as a practical means of controlling certain diseases of sweet potatoes. Poole (*133*), Adams (*3*), and Manns and Adams (*119*) have demonstrated the efficacy of inoculated sulphur in the control of scurf and soil rot. Poole found that 300 to 400 pounds of inoculated sulphur applied broadcast about one month before planting reduced considerably the loss from scurf and soil rot. Adams found that not only were scurf and soil rot greatly reduced by the application of sulphur, but evidence was obtained which indicated that black rot in the field and its development in storage was reduced. He found that 300 pounds of inoculated sulphur was all that could be used safely in Delaware without danger of injury. He demonstrated that sulphur not only reduced the proportion of disease but that it actually acted as a fertilizer. The evidence indicates that inoculated sulphur at the rate of 200 to 300 pounds per acre applied broadcast several weeks before the plants are set out will partially control scurf, soil rot, and possibly black rot. Care must be taken however, not to apply too much sulphur or to apply it too often, since the application of excessive quantities of sulphur will cause injury to

the plants and to the potatoes not only during the year it is applied but during succeeding years.

Crop Rotation

Since plants are often infected in the field, it would be a waste of time, labor, and money to grow disease-free plants and then subject them to the danger of infection by planting them on ground where the disease occurs. It is not known definitely how long the fungi causing the several sweet-potato diseases will live in the soil without their natural host, but it probably is several years. It is not likely that all fungi survive the same length of time in the soil. Neither is the danger from field infection the same for all fungi. Foot-rot infections in the field are few, so that rotation for this disease alone would not be highly essential. However, no place is known where foot rot is the only sweet-potato disease of importance. The organisms causing stem rot, black rot, scurf, and soil rot remain in the soil for several years, so that the rotation should be as long as is consistent with the usual farm practices, but certainly not less than three or four years; and where soil rot is bad a longer rotation is recommended. Any crops common to the district may be used in the rotation, since these fungi are not parasitic to other crops. *Fusarium hyperoxysporum*, one of the stem-rot fungi, has been successfully inoculated into *Ipomoea hederacea*, a common inhabitant of sweet-potato fields. However, diseased specimens of this species have never been found under natural conditions. Several species of Ipomoea have been artificially infected with *Ceratostomella fimbriata*, the fungus causing black rot, but no diseased plants have been found in the field where black rot was present. The sweet potato and a wild species of morning glory, *Ipomoea hederacea*, are presumably both hosts for the white-rust fungus, *Albugo ipomoeae-panduranae*. White rust is rather common in sweet-potato fields in some parts of the world during weather favorable to its development, although in the United States it is seldom severe enough to cause any damage to the crop. From the standpoint of the farmer the species of wild morning glory, therefore, need not be considered as of any particular importance.

Resistant and Susceptible Varieties

The question of varietal resistance and susceptibility is one of considerable economic importance in the control of plant diseases. The introduction, selection, or breeding of resistant varieties and strains is sometimes the only solution of the disease problem. During a period of six years careful notes have been taken of the occurrence of the several diseases in the various sweet-potato varieties. Furthermore, considerable experimental work has been done in testing the resistance and susceptibility of the different varieties to the several diseases of economic importance.

Stem Rot

Some of the commercial varieties of sweet potato (Yellow Jersey, Big-Stem Jersey, Red Jersey, Nancy Hall, Porto Rico, Gold Skin,

and Belmont) are extremely susceptible to stem rot, and a large percentage of the plants are likely to become diseased during the growing season. On the other hand, there are a few varieties (Pumpkin, Pierson, Creola, Dahomey, Southern Queen, Red Brazil, Yellow Strasburg, White Yam, Haiti Yam, Triumph, and Key West Yam) which, when planted on badly infested soil, will give a good crop, the infections, if there are any, being so slight as to cause little or no apparent loss. Harter and Field (*81*) have shown that some of these varieties, while immune under natural conditions, could be infected to a slight degree by artificial inoculation. In such cases the fibrovascular bundles were blackened 1 or 2 inches on each side of the court of inoculation.

More recently Poole (*135*) has tested several of the southern varieties and several strains of the Yellow Jersey on badly infested soils in New Jersey. The results of these investigations showed that White Yam, Red Brazil, and Triumph were almost entirely immune to stem-rot infection and in some cases produced an excellent yield. The different strains of the Yellow Jersey variety and the Nancy Hall and Porto Rico were from 15 to 60 per cent susceptible to the disease. Harter and Whitney (*96*) investigated the relative susceptibility of 21 varieties at two stations in Delaware on naturally infested soil. Among the varieties were several known to be very susceptible to stem rot and others more or less resistant. The collection included most of the varieties grown on a commercial scale in the United States. These varieties were grown for four consecutive years at one of the stations and for two at the other. The results showed that none of the varieties is immune. Among the susceptible varieties the mortality was high the early part of the season, though more plants continued to die throughout the summer and fall. On the other hand, only an occasional plant among the more-resistant varieties became infected or died.

The roots and stems were carefully examined for infection by cutting open the roots with a knife when the crop was harvested. The examination of the plants at this stage of growth revealed the fact that some were mildly infected, but as they did not exhibit any external symptoms they were recorded as not infected. The infection took place through the wound made by pulling the plants from the mother potato, at a soil-rot lesion, or at a wound made by other means. It extended only a very short distance from the point of entrance. Apparently no injury was caused, the plants yielding a normal crop.

Another method for meeting the stem-rot situation has been worked out and recommended by Poole (*138*). By this method no attempt was made to control stem rot but to grow a crop in spite of it. By means of a series of experiments in New Jersey, with susceptible Jersey strains, Poole showed that a normal crop could be grown on badly infested soil by planting two or three plants in a hill instead of one. Whether or not two or three plants should be set in a hill depended on the severity of the soil infestation. He found that one or two plants in a hill would usually escape infection, so that a full stand and normal crop was produced. If all the plants in a hill should escape infection, only one produced potatoes. The disease was not necessarily transmitted from diseased plants to healthy ones

of the same hill. Poole demonstrated that the return per acre by this method exceeded the additional cost of growing and setting two or three plants in a hill. Whether or not this method will solve the stem-rot problem permanently remains to be seen. Theoretically, one would expect the infestation of the soil and the infection of the seed potatoes to increase to a degree where not even one plant out of three would remain healthy and yield a normal crop.

The substitution of one variety of sweet potatoes resistant to stem rot for another in any region can not be done alone upon the basis of their resistance to disease. It is a well-known fact that a variety that is productive in one region is not necessarily so in another. For instance, the Yellow Jersey, which produces potatoes of exceptional quality and shape in New Jersey and Delaware, may produce a very stringy, rooty potato in the South. Furthermore, some of the sweet potatoes very productive in the South, such as Red Bermuda and Florida, yield poorly in the Northern States.

Black Rot

Field data collected for a number of years show the following varieties to be susceptible to black rot: Nancy Hall, Yellow Jersey, Big-Stem Jersey, Red Bermuda, White Yam, Southern Queen, Pierson, Florida, Yellow Strasburg, Key West Yam, Red Jersey, Dahomey, Red Brazil, Yellow Yam, Vineless Yam, Belmont, White Gilke, Pumpkin Yam, Eclipse Sugar Yam, Porto Rico, and Triumph. Natural infections of these varieties have been found in the storage house, as well as in the field or seed bed, or both.

Harter, Weimer, and Lauritzen (*91*) supplemented the above observations by conducting a 4-year test of 21 varieties, including most of those grown commercially. The roots of the young plants were dipped in a spore suspension of the black-rot fungus just before they were set in the field. Many of the plants were killed when quite young, but others survived and produced a crop. When the crop was harvested a few diseased specimens were observed, the percentage of apparently infected potatoes being relatively small. At digging time 1 bushel of each variety was taken just as they came from the field and stored at a temperature and humidity suitable for the development of black rot. At the end of about seven weeks they were removed from storage and observations made on the condition of the potatoes. It was found that during the storage period a large percentage of the potatoes of all varieties bore one or more black-rot lesions. It is evident from these results (1) that, even though there is little evidence of infection when the potatoes are harvested, there may be many infections too small to be seen with the unaided eye which quickly develop when the potatoes are placed in favorable situations; and (2) that, so far as the varieties used in these experiments are concerned, none of them are immune or sufficiently resistant to black rot to warrant continued planting on the same ground.

Foot Rot

The foot rot caused by *Plenodomus destruens* is a disease to which there seems little hope of obtaining resistant varieties. Inoculation experiments have demonstrated that a large number of the common

varieties are susceptible, as the following list will show: Yellow Jersey, Big-Stem Jersey, Pierson, Yellow Strasburg, Red Jersey, Red Bermuda, Southern Queen, Dooley, Yellow Yam, Pumpkin Yam, Vineless Pumpkin Yam, and Triumph. It is not unlikely that all varieties are susceptible to foot rot.

During the summer of 1917 the above-named varieties growing in the field were inoculated with an organism that had been carried in culture since 1912. This strain carried in culture for five years failed to infect, probably owing to the loss of parasitism, although it was exceedingly virulent in 1912 and 1913. During 1917, after the above inoculations were made, a fresh isolation of the organism was made from sweet-potato plants from California. This strain proved to be very parasitic, and the organism was isolated from some of the diseased plants. The season was too advanced for satisfactory field tests, so the varieties listed above were grown in pots of sterilized soil in the greenhouse, and after the plants were well started they were inoculated with the virulent strain by the insertion of spores and hyphae into the stem at about the soil line. A large percentage of all the inoculated varieties developed typical symptoms of the disease in one to two months.

Texas Root Rot

Texas root rot is very destructive to a great variety of crops, including the sweet potato, in Texas, Oklahoma, New Mexico, Arizona, and a few other places. It has been reported from Mexico. So far as the writers are aware no tests have been made of the varietal resistance and susceptibility of sweet potatoes to the Texas root rot, and no effective control measures have been worked out. Shear and Miles (*157*) found that deep fall cultivation reduced the severity of the disease. Clean cultivation and crop rotation with immune crops, such as some of the grasses and cereals, are beneficial. King (*102*) found that the spread of the disease could be arrested by soaking with formaldehyde (1 part of 40 per cent formalin to 100 parts of water) an area 2½ to 3 feet wide outside the diseased spot to a depth of about 4 feet. King and Loomis (*104*) found that the methods recommended for the control of root rot in Texas were not efficacious in New Mexico. They demonstrated a considerable increase in yield by the application of 10 or more tons of stable manure per acre to the soil in New Mexico. They reduced the area affected by the addition of organic materials, such as spoiled alfalfa, to the soil. The disease was not eliminated by the use of stable manure, but the benefits derived from it by the plants seemed to enable them to yield a crop in spite of the presence of root rot.

Scurf

No varieties are resistant to scurf, so far as is known. From data collected over a period of years the following varieties were found to be susceptible: Yellow Jersey, Red Bermuda, Japan Brown, Red Brazil, Florida, White Gilke, Vineless Pumpkin Yam, Pumpkin Yam, Eclipse Sugar Yam, Porto Rico, Triumph, Yellow Yam, Yellow Strasburg, Creola, Belmont, Pierson, White Yam, Key West Yam, Big-Stem Jersey, Nancy Hall, Southern Queen, Dahomey,

and several unnamed varieties. Most of these varieties have been tested by McClintock (*117*), some of which had little or no scurf when grown for a season in Virginia. The writers have found, however, that some of the varieties mentioned by him as having little or no scurf were the most susceptible and worst affected in other parts of the country. It is believed there is little difference in the susceptibility of the different varieties, since varieties which have shown slight infection in one section of the country were the worst infected in another.

Soil Rot

A test of 20 varieties of sweet potatoes for resistance to soil rot, both in New Jersey and in Delaware, showed that in general those with red skins were more resistant than those with white and yellow skins. All of the varieties tried were susceptible to the disease in varying degrees. Big-Stem Jersey, Nancy Hall, Porto Rico, Pumpkin Yam, Dooley, Yellow Jersey, and General Grant Vineless are very susceptible; Red Bermuda, Dahomey, and Red Brazil show considerable resistance. Intermediate between these two groups may be listed such varieties as Pierson, Southern Queen, Yellow Strasburg, Red Jersey, Triumph, Yellow Yam, Florida, and White Yam.

The use of resistant varieties can not be applied generally to the control of soil rot. Although the red-skinned varieties are more or less immune in some parts of the South, there is very little soil rot where they are grown. Where the susceptible white-skinned or yellow-skinned varieties are grown, the red-skinned varieties might be substituted for them. On the other hand, none of the relatively resistant varieties are grown in the northern sweet-potato belt, except the Red Jersey, which has been largely replaced in recent years by the Yellow or Big-Stem Jersey. The growers in New Jersey, Delaware, Maryland, and Virginia, as well as in some of the other States, have established a market in the large cities (like New York, Boston, and Philadelphia) for yellow-skinned potatoes of the Jersey type. In view of this, together with the fact that losses are more or less local, it would seem unwise to attempt the introduction of a resistant variety unknown to the market unless some other decided advantage was to be gained.

White Rust

Observations and notes taken at the Virginia Truck Experiment Station at Norfolk, Va., in 1917, a season in which there was a rather general infection of white rust, showed that practically all the commercial varieties are susceptible to this disease in varying degrees. Table 2 shows approximately the degree of infection of the different varieties on August 27, 1917.

Albugo ipomoeae-panduranae is said to occur on a number of species of Ipomoea. Although no cross inoculations have been made, considerable evidence is at hand which indicates that the organism occurring on *I. hederacea* is different from the one occurring on the sweet potato. *I. hederacea* badly infected with white rust grows abundantly among sweet-potato plants without the latter being infected. In 1918, near Rosslyn, Va., white rust was first noticed on *I. hederacea* in August, and after that time it increased in sever-

ity, but not a single sweet-potato leaf was found infected during the entire summer. These facts suggest that the two organisms are at least biologically different.

TABLE 2.—*Relative susceptibility of sweet-potato varieties to white rust (Albugo ipomocae-panduranae) on August 27, 1917*

Very badly infected	Badly infected	Medium infection	Slight infection	No infection
Red Jersey. Yellow Jersey. Red Bermuda. Nancy Hall.	Creola. Dooley. Big-Stem Jersey. Florida. General Grant Vineless. Pierson. Yellow Strasburg. Red Brazil. Dahomey. Yellow Yam.	Porto Rico. Southern Queen. Eclipse Sugar Yam. White Yam. Key West Yam.	Triumph. Belmont. Pumpkin Yam. Vineless Yam.	Vineless Pumpkin Yam.

No morphological study has been made of the organisms from the different species of Ipomoea. However, the fungi from both *I. hederacea* and *I. batatas* have been examined frequently, and no morphological differences between the two have been noted.

DISCUSSION

Certain varieties of sweet potatoes have been grown continuously in some regions for many years. This continuous cultivation of one or two varieties has enabled the farmers to establish a good market for their crop. For example, the Yellow Jersey, grown largely in New Jersey and Delaware, finds a ready market in the large cities of the East. These markets do not readily take the yam types so generally grown in the South. In the far South, where the yam types are mostly produced, a different market has been established and one which prefers those types to any other. In fact, the markets preferring the southern sweet potatoes do not like the Yellow Jersey or similar varieties and will not accept them if others can be obtained. To substitute the southern varieties for the Yellow Jersey types in the stem-rot-infested soils of the North would mean the loss to the farmer of a profitable industry, or, at least, it would force them to build up a new market for their crop.

There seems to be little hope of producing a strain of sweet potatoes resistant to disease. The manner of their reproduction, that is, vegetatively, would seem to warrant the conclusion that little in the way of results can be expected by selection. The writers selected plants free from stem rot each year for six years, and there was no evidence of increased resistance.

To control or eliminate the several destructive diseases of sweet potatoes successfully requires careful attention to such matters as seed selection, seed disinfection, care in the preparation of the seed bed, and crop rotation (*75*, *133*). If these details are carefully followed at each step the losses can be reduced to a minimum. Undoubtedly one of the most difficult of the diseases to control is stem rot. The writers have learned from experience that the use of what may be called a "breeding plot" is of considerable value in eradi-

cating stem rot and other diseases. While it is advisable to pull up and destroy all diseased plants, it is a big task where the acreage is large. Where it is not possible to do that, it is recommended that the farmer set aside a part of his field from which he will take his seed potatoes for the next crop and rogue out all diseased plants from it during the entire summer. Such a practice does not involve great labor and will greatly improve the crop in a year or two. This method is especially to be recommended if the stems are not split when selecting for seed. It is the custom among farmers to use the second-size potatoes, or those just smaller than the market size, for seed purposes. The diseased plants, if they produce potatoes at all, are more likely to give a large number of potatoes of about the customary size for seed rather than large ones. Therefore, if the seed selection is made on this basis, a large percentage of the diseased potatoes would be used for seed. To prevent diseased plants from producing seed by pulling them up and destroying them would eliminate much loss in the succeeding crop. This practice should be followed each year.

SUMMARY OF CONTROL MEASURES

The preceding discussion of control measures of the field diseases embodies considerable experimental data, which have been presented in detail. For the convenience of those readers who are interested merely in the methods to be employed, the control measures are here summarized briefly by diseases.

Stem Rot

At digging time select seed potatoes from disease-free plants, as determined by splitting the stem from about the soil line into the roots. Disinfect the potatoes before bedding in a 1 to 1,000 solution of corrosive sublimate for 5 to 10 minutes. Use a clean seed bed. Plant in noninfested soil. Rotate with other crops. Grow resistant varieties when practicable. The application of fertilizers is of no value so far as the control of this disease is concerned. The causal organisms do not cause a storage rot.

Black Rot

Select seed potatoes free from black-rot lesions. Selections should be made both in the fall and in the spring just preceding bedding. Disinfect as for stem rot. Bed in clean soil. Plant in noninfested soil and rotate with other crops. Black rot is also a storage disease. Fertilizers do not reduce the percentage of black rot. There are no resistant varieties.

Foot Rot

Infections with the foot-rot organism do not take place to any extent in the field, but almost entirely in the seed bed. It is therefore important to select disease-free seed and disinfect it thoroughly in a solution of mercuric chloride in the proportions given for stem rot. Use an uninfested seed bed, plant in soil free from the foot-rot fungus, and rotate with other crops.

Texas Root Rot

Texas root rot occurs in fields in Texas, Oklahoma, New Mexico, and Arizona. Deep fall plowing will reduce the disease. Clean cultivation and rotation with immune crops, such as some of the grasses and cereals, are beneficial. Soaking the soil to a depth of about 4 feet with formaldehyde (1 part formalin to 100 parts water) just outside the diseased spot will prevent further advance of the fungus.

Scurf

Select scurf-free potatoes for seed. Disinfect as for stem rot. Use an uninfested seed bed. Rotate with other crops. Plant in uninfested soil. Scurf is worst in soils containing a considerable proportion of organic matter, such as stable manure. No resistant varieties are known. Scurf spreads very little or not at all in storage. The application of 200 to 400 pounds of inoculated sulphur broadcast per acre reduces the extent of infection.

Soil Rot

Soil rot, as far as is known, is not communicated from the seed potatoes to the plants produced therefrom. Infections take place in the field. Rotate with crops other than sweet potatoes for several years on infested land. Build up the soil by growing green-manure crops. Inoculated sulphur broadcast at the rate of 200 to 400 pounds per acre reduces the amount of soil rot and increases the yield. Soil rot does not cause any loss in storage.

Sclerotial Blight

Sclerotial blight occurs in seed beds when humidity is high and weather warm. When beds are covered with canvas or sash the blight can frequently be controlled by removing the sash to reduce the humidity. Stirring the soil is also helpful.

STORAGE DISEASES

In the preceding pages it has been shown that there are several organisms that cause both field and storage disease. For convenience in presentation, the storage diseases from the standpoint of control can be divided into two groups, namely, those that cause diseases in the field and in storage and those that cause loss in storage only. To the first group belong black rot, scurf, and foot rot. Soft rot and ring rot occasionally occur in the field, but they can not be considered as field diseases. The control of storage rots will therefore be considered under (1) elimination, (2) digging and handling, (3) the storage house and its management, and (4) fumigation of potatoes in storage.

ELIMINATION

Black-rot and foot-rot infections that originate in the field continue to develop in storage. Scurf, while not increasing to any extent in storage, causes considerable loss from shrinkage and reduc-

tion in market value by the unsightly appearance of the potatoes. Black rot and foot rot spread in the storage house. It is, of course, possible to reduce the loss from these diseases, even though they are present in storage, by the proper management of the house, but it is not possible to eliminate them. The surest way is to eradicate the organism entirely, which can be done by the use of the proper sanitary methods of control; that is, by careful seed selection and disinfection, by the preparation of a clean seed bed, and by crop rotation.

DIGGING AND HANDLING

Every sweet potato is wounded when it is broken from the stem, and it is principally through these and other wounds that most of the storage-rot fungi are able to enter. A point to be emphasized in this connection is that care must be exercised not to wound and bruise the potatoes any more than is necessary during the process of digging and storing. Some potatoes are unavoidably cut and bruised when they are plowed out, and considerable unnecessary bruising frequently occurs during subsequent handling. Instead of being thrown from several rows into one pile, they should be carefully pulled from the vine and laid in a basket or in crates if crates are used for storage. The potatoes should be allowed to dry for an hour or two in the sun before they are taken to the storage house, thereby permitting the dirt to dry and the skin to harden. If the potatoes are stored in bins, they should be poured out of the baskets or crates with great care. Several bins should be filled simultaneously, so as to allow the moisture to escape as freely as possible.

It must not be forgotten that there must be a wound before infection by most, if not all, storage-rot organisms can take place, and that the greater the care with which the potatoes are handled the less the danger of bruising and wounding. Beattie (*14*) recommends that the potatoes be taken from the field in padded baskets and spring wagons; also that the digging be done on a bright, sunshiny, windy day, so that the potatoes may lie exposed to the sun and wind for one or two hours before being hauled to the storehouse.

THE STORAGE HOUSE AND ITS MANAGEMENT

The storage house should be constructed so that the temperature and humidity can be controlled. Directions for the construction of an efficient storage house have been worked out and published by Thompson (*187*).

Before sweet potatoes are stored, the house should be thoroughly cleaned and then disinfected by any one of several methods mentioned below, or it should be whitewashed. The house may be disinfected by spraying once or twice with a solution of copper sulphate to which a little lime has been added to give it a white color, or it may be sprayed with Bordeaux mixture. The application of a coat of whitewash will serve the same purpose. Winter strength of lime sulphur may be effectively used to disinfect the house. A still better method is to disinfect with formaldehyde gas freed by the addition of formaldehyde to potassium permanganate. The proportion should consist of 3 pints of formaldehyde and 23 ounces

of potassium permanganate for each 1,000 cubic feet of space. The permanganate should be placed in deep receptacles, the formaldehyde poured on it, and the house closed immediately. Formaldehyde gas is an irritant and should be kept out of the eyes and lungs; for that reason it is necessary to work rapidly. The house should be kept tightly closed for at least 24 hours. If the house is large, several receptacles in different parts of it should be employed. In order to dry the house out thoroughly a fire should burn slowly in it for a few days before the potatoes go into storage, and also while the potatoes are being placed, so as to drive off the moisture.

When the potatoes are all in the storage house the temperature should be held at 80° to 85° F. for 10 days to two weeks, after which it should be gradually lowered and held during the storage period as near 55° as possible. The higher temperatures during the curing period help to drive off surface moisture. The house thereafter should be carefully watched as to temperature and moisture. If moisture accumulates it should be gotten rid of by opening the ventilators, or, if necessary, by raising the temperature for a day or two.

Sweet potatoes must not be allowed to freeze. However, there is no immediate danger if the temperature drops to nearly freezing for a day or two. High temperatures should not be permitted for a very long time.

Sweet potatoes in storage must not be handled, unless it is intended to market them at once. It is unsafe to pick them over in order to remove the decayed ones. So doing wounds sound ones, which in turn will decay. The decayed potatoes must be left alone.

Wounding caused by handling and by mice and rats is responsible for most of the decay in storage. Rats and mice seek refuge in sweet-potato houses where the temperature is congenial and food plentiful. They nibble many of the potatoes, making wounds through which enter such organisms as *Rhizopus nigricans*, the cause of soft rot. Many infections can be traced to wounds made by the gnawing of mice and rats. They must be excluded if sweet potatoes are to be kept successfully in storage.

FUMIGATION OF POTATOES IN STORAGE

Some farmers have recommended and certain investigators have suggested that the sweet potatoes in storage be subjected to fumigation to protect them against decay. The possibility of success from such a treatment would depend upon whether or not the fungi causing decay could be killed without the potatoes being injured. With these possibilities in mind, the writers undertook a series of experiments in which (1) burned sulphur and (2) gas released from formalin by potassium permanganate were used as the disinfecting agents.

Only certain details of these experiments need be given. A tight container was used, in which sulphur was burned at the rate of one-half and 1 pound to 100 bushels of potatoes in 360 cubic feet of space. Cultures of *Fusarium hyperoxysporum* (the cause of stem rot), *Rhizopus nigricans* (the cause of soft rot), and *Ceratostomella fimbriata* (the cause of black rot) were put into the container, which was kept tightly closed for 24 hours, after which the potatoes were removed and carefully examined for injury. It was found that the

potatoes were badly injured in spots, the extent of injury corresponding to the quantity of sulphur burned. The spots were more or less circular, somewhat sunken, and appeared to be located at places where the potatoes had been injured or bruised. The injury from the sulphur treatment became more conspicuous after a few days. This treatment did not kill the organisms placed in the containers with the potatoes. These results seemed to prove that sulphur could not be successfully employed, since the potatoes were injured at a concentration which did not destroy the fungi. Moistening the potatoes before fumigation did not alter the results.

Similar results were obtained by treating the sweet potatoes with formaldehyde and potassium permanganate. The proportions of chemicals recommended by Stewart and Gloyer (*173*) for potatoes, namely, 3 pints of formaldehyde and 23 ounces of permanganate to 167 bushels of potatoes in 1,000 cubic feet of space, were employed and the sweet potatoes subjected to the gas for 24 hours, after which they were removed and examined for injury. A culture of several fungi, some of them storage-rot organisms (*Rhizopus nigricans*, *Ceratostomella fimbriata*, and *Fusarium* sp.) were placed in the container. The sweet potatoes subjected to this treatment were likewise injured, and the fungi were not killed. The results of these investigations further showed that sweet potatoes subjected to this treatment decayed more rapidly after removal from the container than did sweet potatoes not treated. This was probably due to the fact that the rot-producing organisms gained a foothold through the injuries caused by the treatment.

SUMMARY

This extended account of the diseases of the sweet potato (*Ipomoea batatas* Poir.) is prefaced by a short statement as to the probable origin of the sweet potato, statistics on the yield and crop losses through diseases, and a discussion of the agricultural practices and methods and their relation to the prevalence and severity of diseases in general.

The field diseases of the sweet potato caused by fungi—stem rot, black rot, foot rot, Texas root rot, scurf, soil rot, mottle necrosis, rootlet rot, sclerotial blight, Rhizoctonia rot, white rust, leaf blight, and several minor diseases—are fully considered with respect to the history of the diseases, their geographical distribution and economic importance, the symptoms, and the pathogenicity, morphology, life history, and dissemination of the causal organisms.

The storage diseases that are caused by fungi—soft rot and ring rot, black rot, Java black rot, dry rot, foot rot, charcoal rot, scurf, surface rot, and a few minor diseases—are considered in the same exhaustive manner as the field diseases. When a disease has been described as a field disease and the same one also occurs in storage, only enough of the field symptoms are reviewed to make clear the connection between the field occurrence of the disease and its appearance in storage.

The physiological diseases and those of unknown cause in both field and storage—mosaic, growth cracking, fasciation, intumescence, sun scald, and internal breakdown—are described with regard to their occurrence, extent of damage, and probable cause.

The control of the field diseases is exhaustively discussed under the following headings: Exclusion, eradication, and resistant and susceptible varieties. A summary of the control measures for the field diseases is given. The control of the storage diseases is taken up under the following headings: Elimination, digging and handling, the storage house and its management, and fumigation of potatoes in storage.

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